FOREWORD

It gives me great pleasure in writing a foreword to this monogram of Dr Shantilal J Shah. I saw him doing Cardiac Catheterization and taking part in cardiac conferences at the Montefiore Hospital in New York in 1954. I was much impressed by his knowledge of the technique and interpretations of the subject. When he asked me to write a few words of foreword for this book I have gladly done so.

Time has long past when the speciality of cardiology could be practised with bed side clinical knowledge and a working acquaintance with electrocardiography. The procedures of cardiac catheterization has made it possible to study the deranged physiology to understand the mechanism of various cardiac symptoms and the haemodynamic basis of efficiency of treatment. It is only by understanding the physiologic basis of each and every clinical finding, the practice of bed side cardiology can reach the level of advances in cardiac surgery.

Dr Shah has tackled here the information to be gathered during cardiac catheterization in acquired heart diseases. After discussing the technique of catheterization and the various normal pressures and patterns he gives a detailed discussion of pulmonary wedge pressures. A full discussion of oxygen studies of the arteries and veins and cardiac output then follows. Vascular resistances are considered and methods of estimating mitral valve area in stenosis and regurgitation receive a consideration.

A study of haemodynamic changes in mitral stenosis receives very thorough consideration and forms good bulk of the book. Pulmonary hypertension in mitral stenosis is fully discussed and so also pulmonary vascular resistance and pulmonary wedge pressure in this type of valvular lesion. A similar consideration of mitral regurgitation follows. A consideration of the pathogenesis of various symptoms and signs in mitral disease is followed by an account of catheterization findings in other acquired heart diseases.

I think a proper and detailed study of this monogram would be well worth and would reward the readers. By a thorough study of the book the reader will get a detailed understanding of the haemodynamics of acquired heart diseases and will be able to follow many other articles on the subject. I feel certain that the reader will have the advantage to manage his cases far more intelligently than heretofore.

Dr Shah has done a fine piece of work and I wish him all success in this venture of his in medical journalism of a very specialised type.

MINOCHER B MODY

PREFACE

This small monogram is the result of my experience during the tenure of my fellowship at the Montefiore Hospital New York

The work is designed mainly for those young Indian physicians who wish to take interest in this ever progressive field of cardiac catheterization in acquired heart diseases

The volume includes the Circulatory haemodynamics in normals and its derangements in some acquired heart diseases The views expressed are those of the author based on the evaluation of available literature as well as laboratory studies

To the informed public the procedure of cardiac catheterization was made widely known after the award of Nobel Prize last year to Drs Forssmann Cournand and Richards To the medical students the procedure has been very fascinating and to the cardiologists it has opened entirely new channels permitting greater degree of exactness and certainty of diagnosis

The field of cardiac catheterization is new to both the old and the young cardiologists With the introduction of cardiac catheterization the understanding of cardiac physiology has advanced considerably The analysis of the data obtained through the cardiac catheter in some acquired heart diseases has thrown light on the complex mechanism of the deranged circulatory dynamics of the patient With the splendid progress in cardiac surgery in the last decade the cardiologist is required to state the diagnosis in more exact terms It is not enough to state that the patient has mitral stenosis or mitral insufficiency It is absolutely necessary to assess the degree of mitral stenosis and/or of mitral insufficiency and also to mention the extent to which mitral disease is directly responsible for the patients symptoms The most reliable method of judging the degree of pulmonary hypertension in mitral or any other disease is the direct measurement of the pulmonary pressures through the cardiac catheter In certain other acquired heart diseases like constrictive pericarditis intrinsic myocardial disease tricuspid valve lesions pulmonary valve lesions etc the data obtained by the right heart catheterization is quite characteristic although it may not be diagnostic Lesions of the aortic valve and to some extent mitral valve necessitated the study by the left heart catheterization

In this presentation any discussion on congenital heart diseases has been purposely omitted because the literate in cardiac catheterization

of congenital heart diseases is available in precise forms elsewhere. However it is felt that an adequate literature regarding the cardiac catheterization in acquired heart diseases in a precise form is not freely available and hence this endeavour.

As a well-equipped cardio-thoracic department is established at the J. J. Group of Hospitals and Grant Medical College, it is considered quite in order if such material could be published under the sponsorship of the Journal of the J. J. Group of Hospitals and Grant Medical College.

Cardio-thoracic department
J. J. Group of Hospitals, Bombay 8
April 1958 S. J. Shah

ACKNOWLEDGEMENTS

Many of my teachers and colleagues at the Montefiore Hospital have provided stimulus directly or indirectly in the preparation of this monogram It gives me great pleasure in thanking Dr Raymond E Weston M D Ph D and Dr Doris J W Escher M D to whom this monogram is dedicated They have provided most constructive criticism and guidance My special gratitude is due to them for the permission they have given to use a good bit of the data obtained during cardiac catheterization at the Montefiore Hospital But for their help this work would not have been possible The permission to publish this material by Dr Louis Leiter Medical Director Montefiore Hospital New York, is gratefully acknowledged

I should like to express my deep sense of gratitude to Dr Minocher B Mody for going through the material and writing a foreword The members of the editorial board of the Journal of J J Group of Hospitals and Grant Medical College are to be thanked for sponsoring this publication My special indebtedness is due to Dr J G Parekh the Editor of the Journal of J J Group of Hospitals and Grant Medical College for spending long hours in correcting and editing this book with extraordinary care and thoroughness I must thank Mr R S Diwan on the staff of the Journal for his help in collecting and arranging the advertisements I would also like to take this opportunity to thank the advertisers in this issue for extending their help in spite of unusual delay in the printing

To my wife last but not least my thanks are due for having made tremendous effort in the preparation of the manuscript diagrams and the tabular data There are many other friends whose help in the preparation of figures and proof readings has been very valuable

The staff members of the press States People Private Ltd have always been enthusiastic in doing their best for this job in spite of many other pressing duties

SHANTILAL SHAH

ABBREVIATIONS

A F   Auricular Fibrillation

A S   Atrial Systole

A—V Valves   Atrio ventricular valves

A—V difference   Arterio venous oxygen difference

A—V fistulla   Arterio venous fistulla

B A / BAm / Brach Art mean   Brachial Artery mean pressure

C I   Cardiac Index

C O   Cardiac output

C P   Constrictive Pericarditis

Dias   Diastolic

D F P   Diastolic filling period

E P   Ejection period

FAm   Femoral Artery mean pressure

I C   Isometric Contraction Period

I M D   Intrinsic Myocardial Disease

I R   Isometric Relaxation

I V C   Inferior Vena Cava

L A   Left Auricle

L P A   Left Pulmonary Artery

L V   Left Ventricle

M I   Mitral Insufficiency

M P A   Main Pulmonary Artery

M R A   Mid Right Atrium

M S   Mitral Stenosis

M V   Mitral Valve

M V A   Mitral Valve Area

M V R A   Mitral Valve Regurgitant Area

M V Resistance   Mitral Valve Resistance

N R   Normal Range

P2   Second Pulmonary Sound

P A   Pulmonary Artery

PAm   Pulmonary Artery Mean Pressure

P A R   Pulmonary Arteriolar Resistance

P C   Pulmonary Capillaries

P D   Proto diastole

PW / PWm / Pulm Art Wedge   Pulmonary Artery Wedge Mean Pressure

P V   Pulmonary Vein

Pre op   Pre operative

Pre comm   Pre commissurotomy

Post op   Post operative

R A   Right Auricle

RAm   Right Auricular Mean

R F   Rapid Filling

R S R   Regular Sinus Rhythm

R V   Right Ventricle

R V dm   Right Ventricular diastolic mean

R V sm   Right Ventricular systolic mean

Ry V Ratio   Rate of decline of v wave

S E P   Systolic Ejection Period

S F   Slow Filling

S I   Stroke Index

S V   Stroke Volume

S V C   Superior Vena Cava

Sys   Systolic

T I   Tricuspid Insufficiency

T P R   Total Pulmonary Resistance

T S   Tricuspid Stenosis

V P   Venous Pressure

THE DIAGNOSTIC APPLICATION OF CARDIAC CATHETERIZATION IN SOME ACQUIRED HEART DISEASES

Vol III April 1958 [Supplementary issue

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Part I

CHAPTER I

DYNAMICS OF THE NORMAL CARDIAC CYCLE

General introduction

The events of the cardiac cycle can be best appreciated by tracing the blood flow through the heart in its normal anatomic pathway Because there are no effective valves between the great veins and the atria the blood from the great veins normally flows into each atrial chamber without any impediment During the ventricular systole when the atrio ventricular valves are closed this inflow of blood begins to dis tend the auricles and the pressure in the auricles consequently rises leading to the well known v wave of the venous recordings When the ventricular systole is over the pressure in the ventricles falls below that in the atria and the A V valves open due to the sucking effect towards the ventricular cavities The blood flows rapidly into the ventricles during which the pressure in the atria falls but the flow continues through the atria into the ventricles

The ventricular contraction then builds up pressure into the ventri cular cavities very rapidly in the beginning until the semilunar valves are opened The short period between the closure of the atrio ventricular valves and the opening of semilunar valves is called the isometric con traction period or the isometric contraction Thus is followed by a brief period of ventricular ejection after which the semilunar valves start closing As the ventricles start relaxing the semilunar valves close com pletely by the back pressure towards the ventricular cavities The period of the process of the closure of the semilunar valves is called the proto diastole during which period the ventricles are neither in communication with the aorta nor with the pulmonary artery Then the ventricles start relaxing rapidly with all the valves closed and this period is called the period of isometric relaxation Soon after this there is a pressure gradient between the atria and the ventricles resulting in the opening of the A V valves and the blood rushes into the ventricles That period of atrio ventricular flow during which both the atria and the ventricles are in diastole occupies about 50% of the time of the entire cycle the atrial pressure being slightly higher than the ventricular one Finally towards the end of the ventricular diastole the atrial systole starts and squeezes some blood into the ventricles The whole period during which the blood flows from the atria to the ventricles is called the period of diastolic filling

The right atrium contracts slightly earlier by 0 013 seconds than the left There follows immediately a short interval of 0 016 seconds between the end of atrial systole and the beginning of the ventricular systole The atrial inflow during the entire cycle is continuous

Thus the cardiac cycle is divided into the following phases —

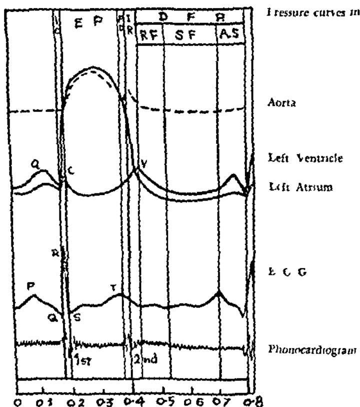


Figure 1

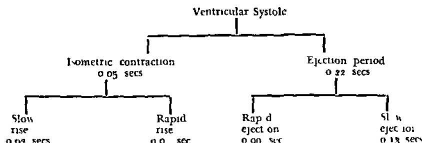
Schematic diagram showing the time relationship between aortic left venticular and left atrial pressure curves with the electro cardiogram and the Phono cardiogram

(Adapted from the Text Books of Physiology by Wiggers Best & Taylor)

I C	Isometric Contraction	D F P	Diastolic Filling Period
E P	Ejection Period	R F	Rapid Filling
P D	Proto diastole	S F	Slow Filling
I R	Isometric Relaxation	A S	Atrial Systole

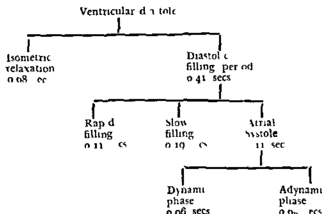
(With the heart rate of 72 per minute one cardiac cycle is 0.8 seconds)

(1)



(2) Proto-diastolic period of 0.04 seconds

(3)



(4) Interval of 0.016 seconds

PHASES OF THE CARDIAC CYCLE IN THE VENTRICLES

Isometric contraction

Following the atrial systole the ventricles are maximally filled with blood. With the onset of the ventricular systole when the pressure in the ventricles exceed that in the atria the A V valves close. The ventricle is a closed chamber and because the contraction of the ventricular musculature on the relatively incompressible volume of blood is not associated with an appreciable shortening of the fibres this is called the phase of isometric contraction. It consists of an initial slower rise of pressure until the A V valves have closed and a later phase of rapid building up of pressure until the aortic or pulmonary artery pressure is exceeded and the semilunar valves open to begin the period of ejection.

The beginning of the isometric contraction coincides in time with the beginning of the first heart sound which follows the Q wave of the E C G by 0.06 seconds. The end of the isometric contraction coincides in time with the apex of the c wave of the atrial tracing.

Ejection period

During the early phase of the ejection the peripheral resistance is less and the arterial tree is relatively less full A great portion of the total stroke output therefore is ejected during this phase As the rate of ejection from this point is much faster than the rate at which the blood is pushed into the venous system via the systemic capillaries the pressure rise in the aorta is relatively rapid This is the phase of rapid ejection After this as the space capacity of the arterial system is diminishing the ventricle has to meet with greater resistance and therefore less volume of blood is pushed out per unit of time thus gradually diminishing the rate of ejection

This is followed by an increase in the speed of shunting the blood from the arterial to the venous system which leads to a situation in the aorta when more blood flows away from the aorta to its branches than is received from the ventricle In the late phase of ejection therefore the pressure falls slightly in the ventricle and the arterial system

Proto diastolic period

After the period of slow ejection begins as the pressure in the ventricles drops below that in the aorta and the pulmonary artery the semilunar valves close producing the second heart sound The duration of incisura on the aortic tracing following ventricular systole forms the proto diastolic period Thus proto diastole is a part of ventricular diastole when the pressures in the aorta and the ventricles are falling before the aortic valves are closed During the proto diastole the blood presumably does not flow in either direction Proto diastole probably represents the duration of the process of the closure of the semilunar valves

Isometric relaxation

Once the semilunar valves close the ventricles are again closed cavities and the phase of isometric relaxation begins Without significant change in the ventricular volume the pressure continues to fall until it is below the atrial pressure and the atrio ventricular valves open Now the atria and the ventricles are again common chambers i e the blood rushes into the ventricles and the atrial v wave descends

The period between the closure of the semilunar valves and the opening of the atrio ventricular valves represents the isometric relaxation During this period a partial vacuum like effect is created within the ventricular chambers At the end of the period the pressure in the atria is high because of the blood accumulated in them during the periods of isometric contraction ventricular systole proto diastole and the isometric relaxation

The end of the isometric relaxation synchronises with the peak of v wave of the atrial tracing In cases of mitral stenosis opening snap of the mitral valve also synchronises with the end of the left ventricular isometric relaxation

Diastolic filling period

With the opening of the atrio ventricular valves the diastolic pressure in the atria (y descent or the downward deflection of the v wave) and the ventricles fall together This is the phase of the rapid filling of the ventricles The blood under pressure in the distended atria rushes in to provide 60 to 80 of the end diastolic volume of the relaxed ventricles The flow during this period depends upon the duration of the diastole the atrio ventricular pressure gradient the area of the atrio ventricular valves and the distensibility of the ventricular myocardium In the succeeding period the phase of slow filling or diastasis ventricular diastolic pressure may rise one or two mm of Hg depending upon the ventricular resistance to its diastolic filling At the end of the diastasis atrial systole forces blood into the ventricles to complete the filling before isometric contraction begins Ordinarily the atrial systole contributes only 10 to 20% of the total diastolic flow but with tachycardia which interferes with the early phase of rapid filling it becomes of greater significance

Rapid heart rate usually reduces the diastolic filling period and this reduction is at the expense of the period of diastasis which serves ordinarily as a period of reserve

PHASES OF THE CARDIAC CYCLE IN THE ATRIA

Atrial systole

After the ventricular diastasis a wave of contraction beginning in the sinus node spreads over the atrial musculature until all of the atrial fibres are in a state of contraction This is called the dynamic phase of the atrial systole during which much of the atrial blood pumping is accomplished This sequence results in the a wave of the atrial systole which has a slowly rising ascending limb reflecting contraction of an increasing number of fibers The symmetrical descending limb of the a wave (X descent) reflects the adynamic relaxation phase of the atrial systole during which contracted atrial fibers relax in a progressive sequence of First to start first to finish Thus the slope of the ascending limb of a wave is similar to that of the descending limb

Normally the atrial a wave begins at the peak of P wave of the E C G and falls entirely within the PR interval of the E C G The peak of a wave precedes the onset of the first heart sound

Highly magnified right atrial activity (giant a wave) is necessary in order to meet with the increased right ventricular diastolic resistance in pure pulmonary stenosis or pulmonary hypertension of varying etiology Well marked a wave is present in pulmonary arterial wedge or the left atrial pressure tracing in cases of long standing mitral stenosis

Atrial diastole

With the onset of the ventricular contraction the rise of pressure closes the atrio ventricular valves and the ventricular isometric contraction begins As the pressure rises in the closed ventricular cavity the leaflets of the atrio ventricular valves are under tension and the bulging of the cusps into the atria produces the sharp c wave of the atrial pressure curve Although the c wave is generally prominent in the pulmonary arterial wedge tracing or the direct left atrial tracings in cases of mitral stenosis due to the forceful closure of the mitral valve it may be inconspicuous in the right atrial tracing because of the gentle closure of the tricuspid valve With the opening of the semi lunar valves ventricular flow begins the tension on the atrio ventricular valves is reduced and the latter regain their original position Thus the c wave falls It is likely that the simultaneous production of negative pressure in the thoracic cavity by ejection of the stroke volume may be a contributory factor to the downward deflection of c wave

With the reduction of the pressure in the atria there is a rapid inflow of venous blood slowly raising the pressure At the point when the blood volume in the atria is maximum the ventricles are isometrically relaxed This corresponds with the height of v wave At the end of isometric ventricular relaxation the atrio ventricular pressure gradient opens the atrio ventricular valves and the blood rushes from the atria to the ventricles resulting in the downward deflection of the v wave (y descent) Following the deflection of the v wave is the beginning of the period of diastasis of the ventricles when both the atria and the ventricles are in a relaxed state The peak of v wave is not higher than that of a wave in normal individuals

A tall v wave in the right atrial tracing is characteristic of tricuspid insufficiency and that in the left atrial tracing or pulmonary arterial wedge tracing is characteristic of mitral insufficiency The gradual fall of the downward deflection of v wave in the right atrial tracing is suggestive of tricuspid stenosis and that in the left atrial tracing is suggestive of mitral stenosis

CHAPTER II

RIGHT HEART CATHETERIZATION

Types of catheters

The catheters are specially manufactured from radio opaque nylon material in standard sizes varying from the smallest No 4 to the largest No 10 The smallest has an internal diameter of 0 5 mm and an external diameter of 1 3 mm while the largest has an internal diameter of 1 8 mm and an external diameter of 3 2 mm No 4 size can be used for infants and No 10 size for persons with very big veins Most commonly used sizes are No 6 7 and 8 The catheters are ordinarily 100 cm long but extra long catheters of 125 cm are also available Double lumen catheters have two openings at the cardiac end namely a proximal and a distal one The outside end also has two openings fitted with Luer lok The cardiac end is bent by means of a stylet to facilitate manouvering inside the cardiac chambers The catheters are somewhat stiff but the pliability is such that it can form a loop

The cleaning of the catheters is very important Proper care on the part of the nurse who washes the catheters after the procedure is over can avoid many pyrogen reactions and hence the failures of the procedures or the dangers to the patient The method for cleaning and sterilization will depend upon the type of the catheter Such instructions are usually supplied by the manufacturers Sterilization may be advised by hot formalin vapour or autoclave Just before insertion the catheter should be flushed with normal saline

Preparation of the patient

All the patients with clinical evidence of congestive heart failure should receive intensive therapy to dry them up to an optimum level The patient has a light dinner on previous evening and a light sedative like barbiturate 1 to 2 grs at bed time Next morning half an hour before the procedure the patient is given morphia 1/4 to 1/6 gr or pethidine 50 to 100 mgm along with scopolamine 1/100 to 1/200 gr

Anaesthesia

In adults with acquired heart disease light sedation is enough to keep them reasonably steady physiologically However in case of younger and unco operative individuals inhalation anaesthesia[2] or parenteral barbiturate or pentothal can be used

Technique

The patient lies in supine position on a radio lucent mattress on the X ray table The right arm is extended to about 90° resting on a side table with a lead shield After an effective local anaesthesia with 1% procaine venesection is performed on the right median cubital or basilic vein The catheter size must be selected to fit the vein 5% glucose solution with heparin (2000 to 5000 units per liter) is dripping through the catheter during the entire procedure at the rate of about 15 to 30 drops per minute in order to prevent any clotting inside the catheter Under fluoroscopy when the tip is judged to be at the required spot the catheter is connected through a two way stop cock to the recording manometer via a statham gauge pressure transducer the whole system being completely free from any air bubble The difficulty that may arise in entering the superior vena cava right ventricle or pulmonary artery can be overcome by rotating the catheter clockwise or anti clockwise and by gentle pushing during the various phases of respiration The pressure tracings are recorded at various places and blood samples are drawn wherever necessary If the venous spasm causes any difficulty it is advisable to lubricate the surface of the catheter outside the vein with heparinized solution especially before any further advance

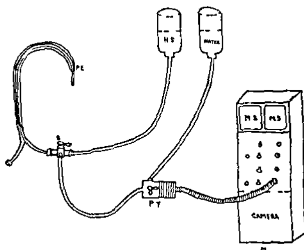


Figure 2

Schematic diagram showing the set up during the procedure of cardiac catheterization

D L C Double Lumen Catheter
D E Distal End
P E Proximal End
S C Two-way Stop Cock
P T Statham gauge Pressure Transducer levelled at zero reference point
H S Heparinized glucose solution running through the catheter
E M Photographic Electro manometer
M S Monitoring Oscilloscopic Screen

Pressure recording system

The pressure recording system consists of pressure transducers and a *recording manometer* At most laboratories the pressure transducers used are statham gauge During the procedure these transducers are levelled to a zero reference point In the present series the zero point was taken to be the junction of the anterior 2/3rd and posterior 1/3rd of the antero posterior diameter of the chest with the patient in supine position Some laboratories take zero point as 10 cms higher from the surface of the bed Wood refers to the sternal angle as the zero point in his works

Three different types of the recording manometers are available —

1 Direct writing multi channel recorders (Sanborn) are used in many laboratories all over the world and are considered satisfactory for routine diagnostic work

2 Photographic electronic recorders (Electronics for Medicine or Atlas) are more sensitive and perhaps more accurate as desired *for research purposes*

3 Jet type electro manometers (Elema) where the stylus deposits a thin jet of special ink on the paper are relatively new in the field but the sensitivity claimed is close to that of the photographic type

Cathode ray monitor (Oscilloscope) is a very useful device to enable the tracings to be watched on the screen continually

Drawing the blood samples

The blood samples for oxygen analysis can be drawn through the catheter from various places in the venous circulation or the pulmonary circulation However in evaluating acquired heart disease it is generally not necessary to draw many samples unless one wants to exclude the congenital heart lesion at the same time For the purpose of calculating the cardiac output pulmonary artery blood is used as a mixed venous blood The blood samples are drawn in heparinized syringes and sealed with mercury Arterial samples are obtained by direct puncture of any of the accessible arteries using Cournand s arterial needle

Complications and their prevention

1 Cardiac arrhythmias —

a Extra systoles—

Premature auricular or ventricular contractions are quite frequent particularly when the tip is in the outflow tract of

the right ventricle The operator need not be worried about it as they disappear by themselves when the catheter tip is removed from the irritating position

b Bursts of supra venticular tachycardia—

This occurs rarely and may lead to shock or pulmonary oedema when prompt treatment is absolutely necessary

c Bursts of ventricular tachycardia—

These are extremely rare and did not happen in any of the patients in the present series If they do occur immediate treatment is required

d Right bundle branch block—

This is occasionally seen in congenital heart disease but is rarely seen in an acquired ones

2 Anaesthetic depression and shock after heavy sedation—
This is not uncommon if the sedation is very heavy However with light sedation it is rare Treatment of shock oxygen etc should be immediately instituted

3 Late post operative fall in blood pressure and respiratory depression

Careful watching of the patient is important until the patient is fully awake This could be missed when the cardiac catheterization is performed without separate staff for it

4 Peripheral emboli—

Blood clot or air emboli may occur if adequate care is not exercised by the operator In cases of associated septal defects the emboli may reach the cerebral or the coronary arteries Thrombo embolism can be avoided by continuous drip of heparinized glucose solution through the catheter

5 Pyrogen reaction—

Pyrogens liberated from the improperly cleaned catheters are more likely to occur when the procedure is continued too long Haemodynamics were considerably altered in one patient in the present series while in the middle of the rigors

Contra indications

The hearts with coronary insufficiency recent or old myocardial infarcts and subacute bacterial endocarditis should form absolute contra indications for the cardiac catheterization Reported mortality rate in Ebsteins disease has been very high[3]

Mortality rate

Cournand et al[1] reported 0 1% mortality in 5691 cases catheterized while Zimdahl[4] reported an incidence of 0 27 in 1000 cases The mortality rate is higher in severely cyanosed children but in adults the mortality is even lower than reported We had no mortality in the present series with the acquired heart diseases

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CHAPTER III

LEFT HEART CATHETERIZATION

The technique of the left heart catheterization is not as much free of risk as that of the right heart catheterization

There are several approaches attempted to reach the left side of the heart each one carrying its own risk

1 Needle approach

a A straight needle is passed into the left ventricle from the apex beat region[7]

b Trans bronchial left atriography consists of puncturing the left atrium through the left bronchus near the carina with a 18 gauge needle 6 cms long attached to a metal tube introduced through a bronchoscope[6,1] We have had considerable experience with this method The procedure of tracing the left atrial haemodynamics through the bronchoscopic needle is relatively simple and harmless

c From the supra sternal fossa a needle is inserted into the left atrium[5]

2 Catheter approach

a A small polyvinyl catheter is introduced through brachial or carotid artery and passed towards the aorta and the left ventricle[8] About 1 c c of the radio opaque material may be injected through it in order to visualize the tip of the catheter

b A polyethelene catheter is introduced through a 20 cms long needle with 1 m m bore inserted directly into the left atrium from the posterior thoracic wall[2,3,4] The catheter then can be manipulated so that it enters the left ventricle through the mitral valve

c A catheter is passed through the trans bronchial needle (as mentioned above in 1 b) into the left auricle and left ventricle

d A catheter can be introduced through a needle inserted from the supra sternal fossa into the left auricle and pushing it in the direction of the blood stream until the pressure tracings reveal that the catheter tip is at the desired place in the left heart

Indications for left heart catheterization

Mainly the usefulness of left heart catheterization lies in the diagnosis of the double mitral and aortic valve lesions

a Mitral lesions The increasing need to determine the predominance of the mitral stenosis over the mitral insufficiency before the patient is submitted to the surgery is being appreciated more and more these days Recent methods of approaching the left heart are considered more useful and reliable On the basis of our experience of the trans bronchial left atriography it is felt that a regurgitant jet will at least be reflected in the left atrial tracing

b Aortic lesions The degree of aortic lesion is better judged by simultaneous tracing in the left ventricle and the aorta

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CHAPTER IV
NORMAL PRESSURES AND PRESSURE PATTERNS

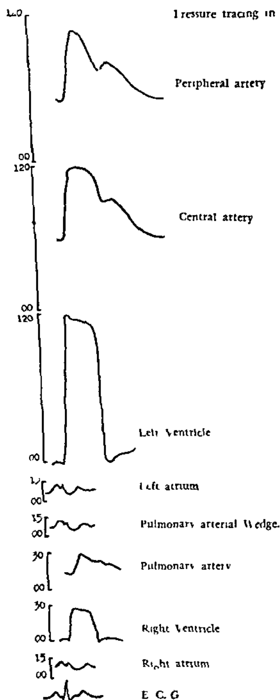


Figure 5
Figure showing the pressure tracing at various places simultaneously with the E C G
(Drawn theoretically)

Mean pressure

Mean pressure in a particular chamber of the heart is the pressure which would have been maintained constantly during the entire cardiac cycle were the pressure not to fluctuate during systole and diastole Mean pressure is arrived at after considering not only the heights of systolic and diastolic pressures but also the time duration of systole and diastole Thus it is not the mid point between the systolic and diastolic pressures as may be commonly supposed

How the mean pressure is arrived at

Before the days of electro manoneter a method of mechanical integration of the pressure tracing taken on a graph paper was used The presentation of the details of this mechanical integration is out of scope of the present work With the availability of electro manometers the mean pressure is obtained by electronic integration

Systolic and distolic pressure

Systolic pressure in the ventricles or arteries is the maximum height of pressure during each cardiac cycle Diastolic pressure is the level of pressure at the base line of the tracing

Curves of the atrial pattern

The tracings of the venous pattern as described previously are obtained only along the venous side close to the heart and fade away further from the heart because the amplitude of phasic variation becomes too small to be recorded due to the damping

The atrial pattern is obtained at the following places —

- i Superior vena cava near the right auricle and to a large extent in the carotid and jugular veins
- ii Inferior vena cava near to the right auricle and to some extent in the hepatic and the renal veins and beyond
- iii Throughout the normal right atrium
- iv Pulmonary arterial wedge
- v Pulmonary veins
- vi Throughout the left atrium

Cyclic phases of the atrial tracing

This has been considered partly in the discussion of the normal cardiac cycle

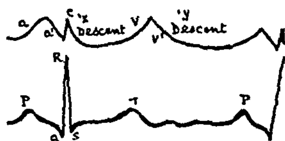


Figure 4

Figure showing the cyclic phases of the right atrial cycle in cardiogram

The a and v waves represent two phases of the atrial systole The ascending limb of the c wave is simultaneous with the isometric contraction of the ventricular systole The peak of the c wave coincides in time with the opening of the semilunar valves with the midpoint of the first heart sound in the phonocardiogram and follows the q wave of the E C G by 0 04 to 0 08 seconds

Unfortunately the a and c waves are not always distinguishable particularly with the tachycardia, when they are superimposed and may appear to be forming one single wave The complexity of all varieties of waves produced in abnormal atrial patterns has not been yet exactly understood but a wave would be affected by the atrial musculature and c wave would be disturbed by the elasticity of atrio ventricular valve cusps The descending slope of the c wave is called the x descent of the atrial pressure curve

Most interesting as a single factor is a production of the v wave The v wave starting during the isometric relaxation of the ventricular diastole is the result of the greater and more rapid inflow of blood into the atria than its outflow into the ventricles Thus the height of v wave *can indi*cate the positive balance of the amount of blood in atria during the isometric relaxation phase of the ventricular diastole If there is any other source of blood entry into the atria previous to the isometric relaxation i e during ventricular systole in mitral insufficiency the amount of blood in the atria during early ventricular diastole would be greater and the v wave would be higher (This point has been discussed later in the consideration of the mitral disease)

The range of pressure in the atria is normally considered to be systolic 4 to 8 mms Hg diastolic O to 4 mms Hg and the mean 2 to 6 mms Hg Taking the atria as o level as mentioned previously)

Left and Right atrial patterns

The right atrial pressure tracing was routinely recorded in all the cases studied by cardiac catheterization We have also recorded left

atrial tracing in well over 60 cases either by direct puncture at the surgery or through the bronchoscopic needle Although both atrial tracings normally are grossly similar the v wave in left atrium is taller has a sharper peak and falls little later than that in the right atrium

CURVES OF THE VENTRICULAR PATTERN

The general pattern of the left and the right ventricular curves are qualitatively similar There are small changes observed in the ventricle in different individuals or even at different places in the same ventricle and during different phases of respiration

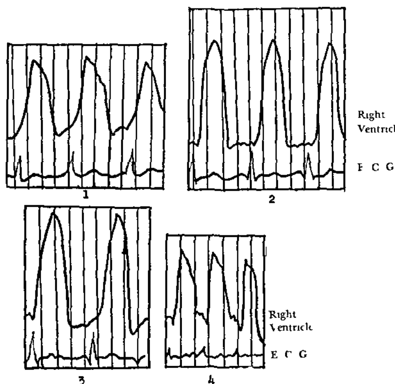


Figure 5

Pressure tracings record at different places in the right ventricular a ity n different ind viduals showing minor variation in the pattern
Each vertical l ne is o seconds apart

5 shows four different types of the right ventricular pressure ained at different places in the right ventricular cavity in uals during the present study Almost all of the right

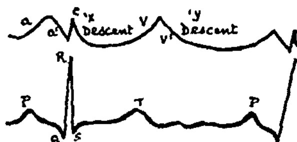


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central arteries This is probably because the distensibility of the pulmonary artery is more than central systemic arteries

The pattern consists of

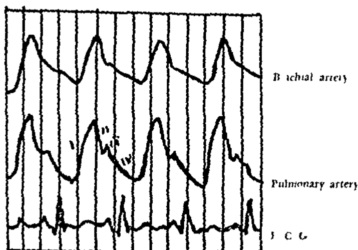


Figure 6

Simultaneous pressure recordings in the brachial artery and the pulmonary artery alongwith the Electrocardiogram
Each vertical line is 0.2 seconds apart

i Ascending limb
ii Pre dicrotic descending limb
iii Dicrotic notch
iv Post dicrotic descending limb

i Ascending limb

ii Pre dicrotic descending limb

iii Dicrotic notch

iv Post dicrotic descending limb

The normal values taken at this laboratory are systolic 23 to 25mms Hg diastolic 8 to 10 mms Hg and the mean of 13 to 15 mms Hg[4, 5] The mean pressures above 18 mm Hg are considered as pathological

ARTERIAL PRESSURE TRACINGS

The catheterization of the left ventricle and the arterial system has more clearly shown the sequence of events occuring in the central arterial system in relation to the left ventricle[11, 12] The normal arterial curve has a progressive shift in pattern from the aortic valve to the peripheral arteries This can be observed in the tracings by the retrograde passage of the catheter up the arterial tree to the aortic arch The cardiac

catheterization of the aorta through the left side of the heart and simultaneous tracing by a needle puncture of an accessible artery such as brachial, radial or femoral can also demonstrate a change in the pressure pattern towards the periphery. The increasing probability of the surgical correction of the aortic valve lesions has aroused great enthusiasm among the physiologists and the cardiac surgeons to understand the physiology of the working of the aortic valve. It is therefore important to consider fully the arterial tracings at all the levels in the arterial tree.

Peripheral arterial tracing

The peripheral arterial wave has been conventionally described as having an ascending and a descending limb with a dicrotic notch and dicrotic wave.

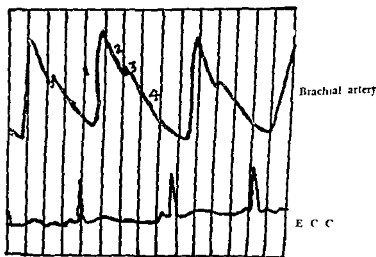


Figure [illegible]

Simultaneous brachial artery tracing and the E C G

1 Ascending limb
2 Pre dicrotic segment
3 Dicrotic notch
4 Post dicrotic segment

Each vertical line [illegible] seconds apart

The ascending limb

The slope of the ascending limb will depend upon an integration the several factors

1 Rate of ejection of blood from the left ventricle

Rate of ejection = Stroke output/Systolic ejection period

Thus the rate of slope varies directly as the stroke output an inversely as the time of the systolic ejection

2 Elasticity of the arterial walls

In the arteriosclerotic vessels partial rigidity of the walls prolongs the systolic ejection period and hence the ascending limb will be prolonged

3 Peripheral resistance

When the peripheral *resistence is low the upstroke will be steep* because of the rapid ejection against low resistance and the amplitude will be large

The descending limb

The descending limb has a pre dicrotic and a post dicrotic segment The pre dicrotic segment represents mainly the slow ejection phase of the ventricular systole and the decent during this period is quite sharp The post dicrotic part represents the beginning of the ventricular diastole and the fall to the peripheral diastolic level is more slow and gradual

The dicrotic notch is a small notch in the descending limb of the arterial curve the downstroke of which is formed by the closure of aortic leaflets The sequence of events leading to the closure is as follows As the ventricle starts relaxing eddie currents beyond the aortic valve appose the cusps At the same time the elastic aortic musculature compresses the column of blood forcing it bi directionally The column of blood moving backward to the aortic valve forces the leaflets down towards the ventricular cavity This sudden descending movement of the aortic cusps supporting a column of blood produces the negative deflection *that is transmitted to the periphery and is seen as the dicrotic* notch This period of formation of the dicrotic notch is called the proto diastolic period Instantaneously after this the aortic cusps are suddenly *released* and partly because of their inherent *elasticity and partly because of the release of pressure by the* blood column abruptly attain their original position The resultant rebound of the blood column sets up a short rise in pressure in the aorta seen as a small positive rebound wave which is transmitted towards the periphery as a positive dicrotic wave which blends into the post dictrotic segment of the curve

Central aortic tracing

The essential features of the aortic pressure pulse near the aortic valve are three contours —

i Ascending limb
ii Rounded contour at the summit
iii Descending limb during diastole

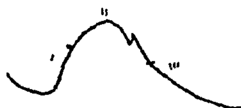


Figure 8
Schematic diagram showing the general contour of the central aortic tracing
i Ascending limb
ii Rounded contour at the summit
iii Descending limb during diastole

The characteristics of this pulse are preserved in transmission to the innominate lower carotid and the subclavian arteries As the pulse is transmitted peripherally however the central characteristics are altered because of the elasticity of the walls damping etc

The fundamental configuration of the aortic tracing may also be affected by several other smaller vibrations external impacts and reflections These can be positive or negative waves and what is recorded is the algebraic sum of all these Minor changes fade away as they travel along the vessels

Arterial pulse wave velocity

It is quite important to have a clear conception of the central arterial pulse and its transmission towards the periphery It has been estimated that the velocity of the blood from the aorta to the arteries of the arm or of the legs is approximately 0 1 to 0 5 meters per second But the rate of pressure wave transmission is 10 to 15 times faster than the blood flow because pressure transmission is effected from molecule to molecule without actual foreward movement of the individual molecules of the blood As the age advances and as the arteries become sclerotic and lose elasticity the speed of the wave transmission becomes faster[15 16 17] Various workers have estimated the speed of the pulse wave Bramwell (1922) estimated it to be 5 2 meters per second at the age of 5 years and 8 6 meters per second at the age of 84 years Schnabel et al found the time interval from the Q wave of the E C G to the onset of the brachial and femoral arterial pulse wave averaged 0 167 and 0 211 seconds respectively As estimated separately by Schnabel et al the speed of the pulse wave was 5 meters per second in the aorta and 8 meters per second in the brachial and the iliac arteries

The pulse wave velocity findings in relation to age were estimated by Hallock as follows —

	Mid childhood Meters/second	22 years age Meters/second	65 years age Meters/second
Aorta	4 1	5 2	10 5
Brachio radial artery	5 1	6 3	9 6

Various other workers have described methods for recording the arterial pressure pulse and pulse wave velocity[11, 12, 13, 1]. The time interval taken by an arterial wave to travel from the aortic valve to the peripheral arteries can be calculated as follows —

Time interval between the onset of Q wave of E C G and the initiation of the arterial pulse in peripheral artery	minus	Isometric contraction period of left ventricle assumed to be the average of 0 05 second	plus	Time lag between electrical and mechanical systole varying between 0 06 to 0 10 second	plus	Time lag in mechanical transmission through the catheter 0 10 second at 37 C Cournand et al 19[illegible]

A time interval calculated as above naturally represents the time interval taken by the pulse wave to travel from the aorta just at the aortic valve towards the point on the peripheral artery under record

Simultaneously recorded pressure tracings in the pulmonary artery brachial artery and the femoral artery along with the E C G are shown below in a case of pulmonary hypertension

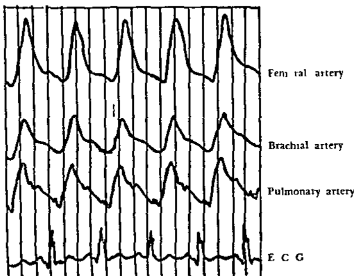


Figure 9

Pressure tracings recorded simultaneously in the femoral artery brachial artery and pulmonary artery along with the E C G in a case of pulmonary hypertension
Each vertical line is 0 2 second apart

In the above figure the time interval between the Q wave of the E C G and the initiation of the brachial artery pulse is approximately

0 23 seconds and the time interval for the femoral artery is 0 27 seconds The calculated approximate speed of the pulse wave in the aorto brachial system therefore is 6 88 meters per second and that in the aorto femoral system is 10 58 meters per second The length of the aorto brachial system roughly is 53 cms and the aorto femoral system is 93 cms

Given below are the figures of the time taken by the pulse wave as calculated from the data obtained by various workers —

Reference	Site	Time interval taken by the pulse wave in normals	Time interval in arterial diseases
Katz et al [10] 1935	Subclavian artery	Immeasurably small	
Wolferth [19] 1935	Carotid artery	Immeasurably small	
Coblentz B et al [3] (3)	Brachial artery	0 02 to 0 04 seconds	0 02 to 0 05 seconds

In hypotension and vaso dilatation due to any cause the pulse wave velocity becomes much smaller and in hypertension perhaps the speed becomes much faster [9]

Changing contour towards the periphery

The dimensions of the arterial pulse vary increasingly towards the periphery Several complex physical phenomena[14] going on at the same time change the contour entirely by the time it reaches the periphery To and fro movements of the coloumn of blood near the aortic valve propulsive driving of the blood by the muscular arterial wall etc are mainly responsible for the changing contour

Aorto femoral system

A series of tracings are shown in the figure 10 as one goes away from the aortic arch towards the bifurcation of the external iliac artery

The following changes should be noted in the aorto femoral system —

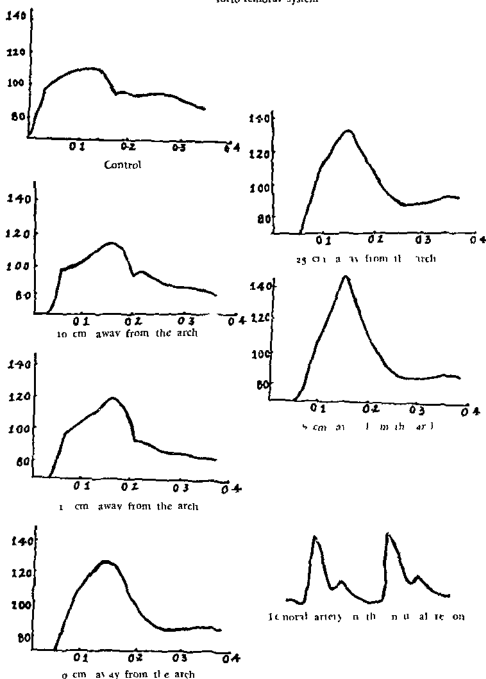
i Progressive delay in the initiation of pressure

ii Alteration in form

iii Rise of systolic peak

iv Mean systolic pressure remaining the same

v All peaks fall nearly at the same time



Figure

(Adapted from Hamilton and Dow Am J Physio 12 48 1939)

There is a propulsive movement of fluid in the tube system and therefore one part accomodates a coloumn of blood which a moment later is accomodated by the subsequent one This when visualized in a continuous stream will show acceleration and deceleration alternately During systolic ejection therefore the proximal and the distal ends of the aorto femoral system accommodate alternately each at the expense of the other an excess of blood As can be easily seen from the curves comparatively more blood is accomodated at a higher aortic system during the first half of ejection and lower aortic system during the latter half of ejection

It is interesting to derive from the curves that the energy is neither gained nor lost as the area covered by the systolic ejection curve is always the same and hence the mean systolic pressures are the same[18]

Aorto radial system

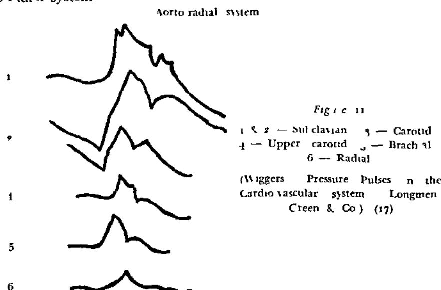


Figure 11
1 & 2 — Subclavian 3 — Carotid
4 — Upper carotid 5 — Brachial
6 — Radial

(Wiggers Pressure Pulses in the Cardio vascular system Longmen Green & Co) (17)

In the aorto radial system as one goes away from the subclavian artery towards the periphery the aortic contour is altered increasingly The ascending limb becomes more and more steep The anacrotic halt of the normal aortic pulse fades as it progresses down from the subclavian It can be seen that by the time the pulse wave comes to the radial artery the preliminary vibrations producing anacrotic band may have disappeared

Measurements of various phases of the cardiac cycle

There are several ways of measuring the period of systole and diastole —

1 Phono cardiography The distance between the beginning of the 1st and 2nd sound can give fairly exact time of duration of systole

2 Pulse tracing of the peripheral artery The distance between the beginning of the pulse and the dicrotic notch measures the systolic ejection period The right ventricular and the pulmonary artery tracing may also be used to measure the systolic ejection period

3 Electro cardiogram The distance between the Q wave of the E C G and the end of the T wave gives systolic ejection period This measurement is not very reliable and it differs in abnormal patterns

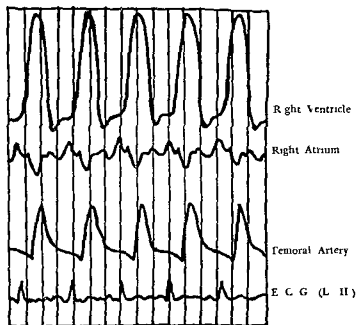


Figure 1

Pressure tracings recorded simultaneously in the right ventricle the right atrium and the femoral artery alongwith the E C G Showing the time relationship in a cardiac patient

Each vertical line 1 o s cond apart

Figure 12 shows the simultaneous tracings of the femoral artery the right atrium the right ventricle and the E C G The total duration of the systole and the systolic ejection period can be calculated easily from these recordings

Normally a linear relationship exists between the total systole the total diastole and the total cycle length

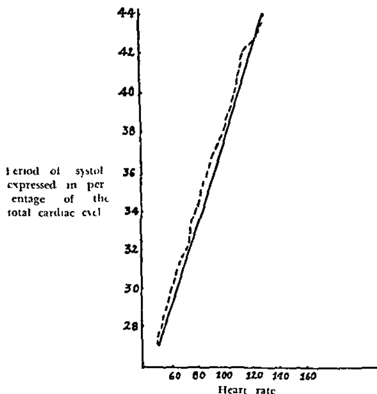


Figure 13

Graph showing the heart rate plotted against the period of systole expressed in percentage of the total cardiac cycle The relationship is almost linear

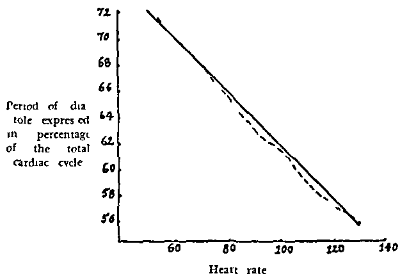


Figure 14

Graph showing the heart rate plotted against the period of diastole expressed in percentage of the total cardiac cycle The relationship is almost linear

The curves above show the relationship of the heart rate with the total systole and the total diastole in a normal person

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CHAPTER V

PULMONARY ARTERIAL WEDGE PRESSURE
THE TECHNIQUE OF OBTAINING IT
ITS GENESIS AND FALLACIES
FACTORS REGULATING THE PULMONARY ARTERIAL WEDGE
PATHOLOGICAL CHANGES IN THE PULMONARY BED AFFECTING THE WEDGE

Pulmonary arterial wedge pressure

There are several terms used in the literature in the place of the pulmonary arterial wedge pressure such as Wedged catheter pressure Pulmonary artery wedge pressure Impacted small artery pressure or Pulmonary end pressure

The technique of obtaining it

It is necessary for those who are not familiar with the procedure of cardiac catheterization to clearly understand how the wedge pressure is obtained As the catheter is pushed from the main pulmonary artery towards the secondary and tertiary branches of the right or left pulmonary artery there is hardly any change as regards the height or the shape of the curve Finally the tip of the catheter is actually wedged into one of the arterioles This jamming of the tip of the catheter into the vessel produces a distal fluid tight compartment with the still smaller branches of the arterioles and their pulmonary network The latter in turn are directly connected with the pulmonary veins and through them with the left atrium

All technical conditions being ideal the pulmonary arterial wedge tracing obtained in this manner is a very close reflection of the pressure changes during a cardiac cycle in the pulmonary veins and the left atrium[7, 1, 12, 3] This is particularly true in cases of pulmonary hypertension of the post capillary origin such as in mitral valvular disease left ventricular failure etc In the present study in some patients the left atrial tracing taken trans bronchially and from the opened chest during mitral surgery compared well with the pulmonary arterial wedge pressures taken during cardiac catheterization several weeks prior to the surgery If there is no difference observed in pressure tracings from the left atrium and the pulmonary arterial wedge one may perhaps assume that —

1 There is no measurable kinetic energy lost during the transmission of pressure waves from the left atrium to the tip of the catheter

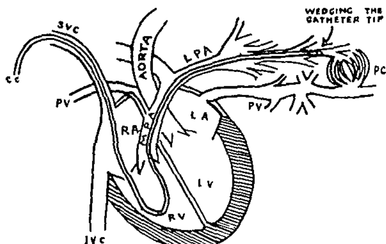


Figure 15

Schematic diagram showing the technique of wedging the tip of the cardiac catheter in a branch of pulmonary arteriole

C C	Cardiac Catheter	L A	Left Atrium
S V C	Superior Vena Cava	M P A	Main Pulmonary Artery
I V C	Inferior Vena Cava	L P A	Left Pulmonary Artery
R V	Right Ventricle	P V	Pulmonary Vein
R A	Right Atrium	P C	Pulmonary Capillaries
L V	Left Ventricle		

ii There is no constriction occurring in the pulmonary veins to affect significantly the pressure system

iii Both the left atrium and the pulmonary veins behave as a single chamber and their elasticity property remains unchanged during the record

Genesis of the pulmonary arterial wedge

It was in 1948 when Hellems and her associates [11] demonstrated in dogs and later in man that the mean pressure recorded through the catheter by the wedging of its tip tightly into one of the pulmonary end arteries closely approximated the left atrial pressure In 1949 Langerlof & Werko[13] showed that the pulmonary arterial wedge pressures are reflections of the left atrial pressure waves occuring during the normal cardiac cycle and of an abnormal left atrial pressure These authors also first pointed out the atrial type of the pulmonary arterial wedge curve in mitral valvular disease

Although the original intent of wedging the tip of the catheter into one of the pulmonary end arteries may have been to measure the pulmonary capillary pressures it is now generally agreed that the entire pressure com

ponent recorded is originated from the left atrium [3,4,5,7,8,10,0,23,24] Similarly when the tip of the catheter is wedged in pulmonary veins it reflects with reasonable closeness the amplitude and the pressure pattern of the pulmonary artery[8,19]

The channel from the tip of the catheter wedged this way to the mitral valve can be divided into two parts —

1 Wedged area of the pulmonary end artery to the arterial side of the pulmonary capillaries

2 Left atrium and the pulmonary veins to the venous end of the pulmonary cappillaries

1 The following factors support the belief that the pulmonary arterial wedge truly represents the venous side of the pulmonary capillaries

a When the pulmonary arterial wedge blood is drawn through the catheter it is found to be 100% saturated It must be assumed that it is drawn by suction in a retrograde fashion from the pulmonary capillaries where the blood is presumably in the state of equilibrium with the alveolar air The fact that it is 100% saturated — not contaminated by blood of a lower saturation escaping from the pulmonary artery beyond the wedged tip—is further proof of a fluid tight system

b There are neither anatomical nor physiological valves in the pulmonary end arteries or pulmonary capillaries As there is a free retrograde flow there can be free transmission of pressure from the pulmonary capillaries to the wedged artery Thus the pulmonary end arterial pressures as recorded through a tightly wedged catheter are a close approximation of the pulmonary capillary pressures However if one wants to be very exact a negligibly small fraction (may be less than 1 mm) might get lost during retrograde flow

c When the pulmonary artery is occluded by a rubber baloon[21] attached proximal to the tip of the catheter the pulmonary arterial wedge tracing shows typical venous type of the tracing which did not change on inflation or deflation

2 Left atrial pressure reflections appear to be transmitted without change to the venous side of the pulmonary capillaries

for the same reason namely the absence of any valve Hence a continuous unobstructed column of blood is present from the mitral valve to the venous end of the pulmonary capillaries

Most observers have agreed that because of the fallacies in the technique of recording the pulmonary arterial wedge pressures it can be taken only as a close approximation of the left atrial pressure within the limits of safety provided the following criteria are satisfied —

i Nature of the tracing is that of an atrial type

ii There is no evidence of damping

iii The tracing in no way should resemble pulmonary arterial tracing

iv Fluoroscopic control demonstrates that during pulmonary arterial wedge recordings the tip of the catheter is pushed peripherally as much as it can go and is free of oscillations

v Oxygen saturation of the blood drawn is 100% except in pulmonary diseases where it may be less than 100%

Fallacies of the pulmonary arterial wedge pressure

1 A length of the catheter passes through the pulsating chambers of the right heart and the pulmonary artery The pulmonary arterial wedge tracing therefore is likely to be distutrbed by these extraneous vibrations

2 Changes during respiration in intra thoracic pressure are likely to produce similar changes in the pulmonary circuit pressures

3 The narrowness of the pulmonary capillary lumen might damp out the pressure transmissions from the left atrium But the enormous number of capillaries available and the dilatation of individual capillaries in mitral valve defects may compensate for any such damping effect

4 Smaller fluctuations from the left atrium are likely to be extinguished in the passage through the pulmonary capillaries and the inhibition of waves can occur in the passage from the pulmonary capillaries to the impacted tip of the catheter

5 Pressure transmission from the venous side of pulmonary capillaries to the arterial side of the pulmonary capillaries might be adversely affected by varying degree of lung distension although this point is highly equivocal

6 Direct impacts transmitted by a hyperdynamic myocardium may disturb the recordings

7 Technical difficulty in determining the zero level

8 Natural decompressive arrangement by means of pleuro hilar veins in cases of pulmonary hypertension of post capillary origin such as advanced mitral stenosis [9]

9 Influence of the bronchial arteries although there is no evidence for it [15, 17]

Factors regulating the pulmonary arterial wedge

(Factors regulating the pressure behaviour in the left atrium in mitral stenosis)

In the above discussion it has been established that the pulmonary arterial wedge is derived from the left atrial pressure which in turn depends on the following factors —

i The degree of mitral valve defect

ii Left ventricular diastolic pressure

iii Volume of blood in the left atrium and the pulmonary venous compartment

iv Changes in the volume elasticity properties of the walls of this compartment

The detailed discussion of the relation between the volume and the elasticity properties of the left atrium and pulmonary venous compartment is beyond the scope of this presentation It can be seen however from a volume elasticity curve given in figure 16 that at lower pressure levels a greater rise in volume of blood in the left atrium can produce smaller rises in pressure while at higher pressure levels a smaller rise in volume can produce greater rises in pressures

As shown in figure 16 x c c of rise in the volume of blood in the left atrio-pulmonary venous compartment can raise the pressure from 5 mm Hg to 12 mm Hg (lower limit of normal to higher limit of normal) Almost equal rise in volume x c c can raise the pressure from 12 mm Hg to 35 mm Hg (from the upper limit of normal to the pulmonary oedema level) About 1/3rd of x c c rise in volume (namely x c c) can raise the pressure from 35 mm Hg to 52 Hg (from assumed pulmonary oedema level to the highest recorded at this laboratory) Pressure volume curves of a similar nature have been recorded by Little [8] in 1949 both in the left and right atria

It follows from figure 16 that in patients with severe mitral stenosis and thickened inelastic left atria with walls stretched to the limit of distensibility a very small additional volume of blood in this left atrial and

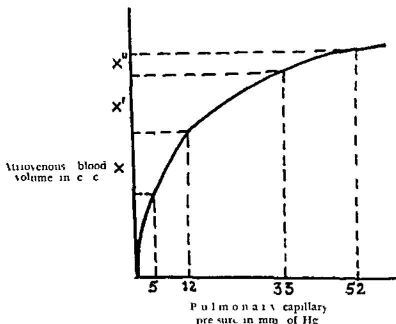


Figure 16
Volume elasticity curve of the left atrio pulmonary venous compartment Theoretical pressure volume curves of pulmonary veins and the left atrium (10)
(Adapted from R Gorlin et al 1951)

pulmonary venous compartment raises the pulmonary arterial wedge pressure to the critical level Thus the pulmonary arterial wedge pressure rises very steeply for a relatively small rise in volume and these patients have a narrow range of safety before pressure levels producing the pulmonary oedema On the other hand in patients with severe mitral stenosis and a thin highly elastic left atrium with quite distensible walls a larger volume of blood may accumulate in the left atrial and pulmonory veins compartment before the pulmonary arterial wedge pressure reaches the critical level

In the first group with a narrow margin paroxyamal dyspnoea and the paroxysmal pulmonary oedema can more easily be produced by smaller variations in the left atrial blood volume while in second group with a wider margin these symptoms are less frequent As the elastic properties of the left atrial walls and blood volume in the left atrium vary from patient to patient the size of the left atrium seen on fluoroscopy is in no way an indication of pulmonary arterial wedge pressure functional inability of the patient or the severity of the mitral valve disease

Pathological changes in pulmonary bed affecting pulmonary wedge -

Although virtually all patients with severe mitral valve disease will have significant pulmonary hypertension less than 40%[14 18] have been mild

or advanced organic changes in the lungs Thus pulmonary sclerosis only occurs in association with marked pulmonary hypertension in mitral disease However except in cases of primary pulmonary disease or congenital heart disease associated with pulmonary hypertension mitral valvular disease remains as the most common cause of pulmonary hypertensive vascular changes

The problem of pathological changes in pulmonary vessels and the alveolar basement membrane in severe mitral stenosis has been studied by Parker and Weiss [16] and later by Larabee et al [14]

The principle microscopic changes in the vessels are described as —

a *Engorgement and dilatation of pulmonary capillaries due to the congestion and an increase in the number of functioning pulmonary capillaries*

b Hypertrophy of the musculature in the arteriolar wall media as in primary pulmonary vascular disease

c Intimal thickening probably due to the fibrous tissue or the hypertrophy of muscular tissue

d Interstitial deposition of collagen in the thickened media and rarely in the adventitia

e A change in the capillary epithelium to the cuboidal type

f Hyperplastic arteriolosclerosis

g Arteriolonecrosis

Quantitative studies of organic changes by Larabee [1] 1949 showed that the patients with only medial hypertrophy have a larger lumen than patients with both medial hypertrophy and intimal thickening

The changes in the alveoli are —

a Congestion and dilatation of capillaries as mentioned above with herniation of capillaries

b Thickening of alveolar basement membrane in 75% of cases

c *Rarely an alveolar epithelial change to the cuboidal*

There is no good correlation found between the severity of any of the above pathological changes and the height of the pulmonary arterial wedge pressures One can assume with impunity that the onset of intimal thickening in the arteriolar walls is a fair indication of high pulmonary arterial wedge pressures of some duration But the converse is not true as the high pulmonary arterial wedge pressures of long duration have

failed to produce pathological changes in pulmonary arteriolar walls of any consequence

Mention may be made here of the fact that there is no correlation existing between the degree of organic pulmonary changes and the severity of the mitral valve defect

Conclusion

In spite of the differences the available experimental and clinical data suggest that the pulmonary arterial wedge pressures recorded through the wedged catheter do reflect left atrial haemodynamics under most circumstances in mitral valvular disease Also if the pulmonary arterial wedge pressure tracing is well marked resembling venous type existence of the nature of mitral valve defect can be confirmed However these reflections in the pulmonary arterial wedge tracing have been somewhat less reliable in the presence of atrial fibrillation Indeed when the patient is fibrillating it may be hard to judge the degree of mitral stenosis or regurgitation even from the tracing taken from the left atrium in the opened chest or through the trans bronchial needle

In some cases of double mitral lesions with auricular fibrillation pulmonary arterial wedge tracing has failed technically to give any further information regarding the predominance of one mitral lesion over the other In that case even surgeon s finger palpating the mitral valve may fail to give any definite information

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CHAPTER VI

METHODS OF ESTIMATION AND SIGNIFICANCE OF CERTAIN DETERMINATIONS

1 Air ventilation air ventilation index oxygen uptake oxygen consumption oxygen consumption index
2 Arterial blood oxygen content capacity and saturation
3 Mixed venous blood oxygen content capacity and saturation
4 Arterio venous oxygen difference
5 Cardiac output cardiac index
6 Methods of determination of cardiac output

Basic to an understanding of cardio vascular dynamics in health and disease is the knowledge of both flow and pressure of blood in the various parts of the heart and the circulation In recent years the technique of cardiac catheterization has permitted the measurement of pressure and flow in various chambers of the heart The determination of the cardiac output in any decompensated heart disease is one of the most important factors to substantiate the clinical picture

In order to be able to calculate the cardiac output various measurements have to be made

1 Determination and significance of air ventilation and oxygen consumption —

These are correctly measured by one of the two methods —

i Direct method

The patient breathes either the room air or compressed air through a closed circuit for an accurately measured period of time The composition of the inspired air is confirmed by its analysis All the exhaled air is collected in a Tissot spirometer in which the volume can be measured after correcting for temperature and barometric pressure The minute ventilation of air is calculated and from this the ventilation index Samples of air are transferred from the Tissot spirometer or Douglas Bag to the tonometers and stored over mercury until analysed for carbondioxide and oxygen by a gas analysis apparatus such as Scholander Hamilton Haldane Van Slyke Electronic etc etc The sum of the percentage of carbondioxide and oxygen subtracted from 100 equals the percent of nitrogen in the expired air

The percentage amount of oxygen in the inspired air given to breathe is corrected by the fraction to obtain the exact percent

$$\frac{\text{Percentage of nitrogen in the exhaled air}}{\text{Percentage of nitrogen in the air given to breathe}}$$

of oxygen supplied to the alveoli for the exchange of oxygen. As nitrogen is an inert gas, the changes in nitrogen are assumed to be the changes in oxygen in the outside breathing circuit and upper respiratory tract and hence nitrogen is used for the correction factor. From these figures the exact amount of oxygen in the inhaled air and the exhaled air are known. The difference is the percent of oxygen taken up by the blood in the pulmonary circulation and is expressed as c.c. per litre of air ventilated. This figure multiplied by the litres of air ventilated per minute equals the oxygen consumption expressed as c.c. per minute. As the air ventilation and the oxygen consumption depend upon the surface area of the patient, they may be expressed in terms of standard reference. The air ventilation in litres per minute per sq. meter of the body surface area is called the air ventilation index. *The oxygen consumption index is similarly expressed.*

It is hardly necessary to emphasize the necessity for a physiologically steady state during the procedure of the measurement.

Example

Suppose a patient with a surface area of 1.3 sq. meters has ventilated 30 litres of air in 6 minutes and the correction factor for the temperature and barometric pressure from the standard tables is 0.980. The correction factor in the standard tables are calculated as

$$\frac{\text{(Barometric Atmospheric pressure—Water Vapour Pressure)}}{760} \times \frac{273}{273/\text{Temperature of exhaled air}}$$

The minute ventilation is $(30/6 \times 0.980) = 4.90$ litres

Therefore the air ventilation index is $\frac{4.90}{1.3} = 3.77$ L/min/sq. meter

Scholander gas analysis is

	Inhaled air	Exhaled air
Oxygen	19.8 %	16.9 %
Carbon dioxide	0.5 %	3.1 %
Nitrogen	79.7 %	80.0 %

The correction factor is

$$\frac{\text{Percentage of nitrogen in the inhaled air}}{\text{Percentage of nitrogen in the exhaled air}} = \frac{80.0}{79.7}$$

Therefore the amount of oxygen in the alveolar air that has taken part

in the oxygen exchange is $19.8 \times \frac{80.0}{79.7} = 19.88$

Therefore the amount of oxygen in the inhaled air is 19.88
and the amount of oxygen in the exhaled air is 16.90

The amount of oxygen taken up in pulmonary circulation is 2.98 volumes % or 29.8 c.c. per liter of air ventilated.

Therefore the total amount of oxygen taken up in the pulmonary circulation per minute of ventilation is equal to

29.8 c.c./litre × 4.9 litres/min = 146 c.c./min

This is the resting oxygen consumption per minute

The oxygen consumption index in this patient therefore is

$\frac{146}{1.3}$ = 112 c.c./min./sq. meter

ii. Indirect method

With the patient breathing through a mouth piece in a closed circuit a record of movement of air in and out of the lungs is obtained by means of the spirometer. The volume of air in the closed circuit is diminished by the amount of oxygen taken up by the lungs during each breath, the expired carbon dioxide *being absorbed by a canister filled with Soda lime in the spirometer.* The slow and steady decrease in the volume of the circuit is recorded on the spirogram by the uniform upward slope of the base line of the tidal air excursions. The oxygen consumption per minute is calculated from the calibration on the recording paper standardized against known volumes after correcting for the *temperature and barometric pressure*.[33]

It should be mentioned however that the indirect method is less accurate than the direct one.

Significance

For cardiac output determinations meticulous care and a high degree of accuracy in measuring the oxygen consumption is required. During exercise or excitement[18, 50, 51] there is a rise

of tissue demand for oxygen which normally is met by an increase in cardiac output

2 Arterial blood oxygen content capacity and saturation

The oxygen in the blood is chiefly combined with the haemoglobin of the red blood cells with about 0 3 c c being dissolved in the plasma of 100 c c of the whole blood The oxygen content is ordinarily expressed in volumes percent or the c c of oxygen contained in 100 c c of blood The oxygen capacity varies directly with the concentration of haemoglobin 1 gm of which combines with 1 33 c c of oxygen Under normal circumstances it may be expressed by the number of c c of oxygen contained in a 100 c c of blood fully oxygenated by exposure to room air The oxygen saturation of the blood is its content expressed as a percentage of its capacity i e the percent saturation is equal to
content/capacity x 100 = % staturation

If the capacity of the blood to hold oxygen is 20 c c per 100 c c and its content 19 c c then that blood is 95% saturated If in a polycythemic patient the capacity is 25 c c and its content 23 75 c c then that blood is also 95% saturated Similarly in a case of polycythemia with a capacity of 24 volumes % even if the oxygen content of the arterial blood is higher than normal namely 21 volumes % in terms of capacity it is unsaturated (only 87 5%) In anemia on the other hand the saturation may be 100% although the capacity may be as low as the content of the normal venous blood i e 14 to 15 volumes % The capacity in cases of polycythemia or anemia, primary or secondary would be high or low respectively The highest observed in our experience is 28 4 vols

Some laboratories prefer to report the blood oxygen content as a % saturation because it expresses the content in terms of the capacity It is believed however that the volumes % content and capacity are a better way of expressing the oxygen values of blood The determinations at this laboratory were done by analysing the bloods by Van Slyke s manometric apparatus by the technique described by Van Slyke and Neil[14]

The percent saturation of oxygen can be obtained directly by the spectrophotometric or oximetric recordings Such instruments unlike gas analysis record only the oxygen combined with the haemoglobin and disregard the amount dissolved in the plasma (0 3 c c as mentioned before) It is extremely important therefore to clearly understand the relationship between the directly read oxygen saturation on the oximeter or photometer and on the Van Slyke s apparatus

The lower limit for normal arterial oxygen saturation is 94% In the absence of pulmonary oedema or other complications the patients

with marked cardiac decompensation do not necessarily show diminished arterial oxygen saturation. When present[3] the reduced oxygenation in uncomplicated cardiac patients usually does not fall below 85%. During exercise little change occurs in the arterial oxygen saturation from the resting saturation despite decompensation unless associated with significant pulmonary disease. The arterial oxygen saturation may even rise slightly due to the increased ventilation. All decrease in a cardiac patient below 94% is important because in that range of dissociation curve smaller changes in saturation corresponds to higher changes in tension.[3, 4, 16]

When arterial unsaturation is present in an acquired heart disease without a shunt, it may reflect inefficient respiration, impaired mixing in the lungs, oedema of the alveolar walls and some times as described in mitral stenosis organic changes in alveolar epithelium.[6] The tachypnoea of the decompensated cardiac patient can also lower arterial oxygen saturation.[39, 4]

Normally incomplete saturation of the arterial blood may result from the following causes —

1. The partial pressures of oxygen in different parts of alveolar system vary. Alveoli having higher partial pressure than 100 mm may not raise the saturation of haemoglobin in the blood any more while alveoli having partial pressure less than 80 mm cannot oxygenate the blood to the fullest extent.[45]

2. Even if the blood is 100% saturated in alveolar capillaries by the time it reaches the left heart it is slightly diluted by blood from the bronchial veins and some of the thebesian veins draining a part of the myocardium.

3. Unlike carbon dioxide blood in alveolar capillaries may not be in complete oxygen equilibrium with the alveolar air or want of adequate speed diffusion across the membrane separating blood and air.[6]

Although the causes of incomplete saturation of the arterial blood are known it has been very difficult to establish any correlation between the degree of decompensation and the arterial saturation. Nevertheless in cardiacs having an arterial saturation below 90% severe disability is present but the converse is not true i.e. the patient having severe dyspnoea or othopnoea may have normal or only slightly reduced arterial oxygen saturation.[15, 29] During exercise there is no reason to expect any drop in the peripheral arterial oxygen saturation except in cardiacs going into pulmonary oedema. Indeed slight improvement in oxygenation may occur because of the increased ventilatory excursion.

3 Mixed venous blood oxygen content, capacity and saturation

Normally the right heart blood at rest is 75% saturated or contains about 14.5 vols of oxygen. The capacity of venous blood is the same as that of the arterial blood. Diminished oxygen in the right heart blood during cardiac decompensation is constantly found [0 31 3]

In a decompensated cardiac patient the tissues have to satisfy their need for oxygen from whatever amount of blood that is circulating through them. The extraction ratio per unit volume of the diminished blood supply is therefore raised. Thus with the reduction in cardiac output during cardiac decompensation marked changes may occur in the mixed venous blood.

In a compensated cardiac patient myocardium has a very poor reserve power and therefore when the oxygen need of the tissue increases during exercise the cardiac output does not rise proportionally. Here the only way for the tissue to get more oxygen is to derive even more of it from the limited volume of blood that is flowing through it[53]. Thus if the myocardium is not able to raise its output proportionally to the elevated oxygen need naturally the mixed venous blood has to give up proportionately that much more oxygen.

4 Arterio venous oxygen difference

The tip of the catheter is kept in the main pulmonary artery all the time when the patient is breathing through a closed circuit for determination of oxygen consumption. The blood samples are drawn simultaneously from an indwelling needle in one of the accessible arteries and from the pulmonary artery through the catheter. The oxygenated arterial blood represents that going to the peripheral tissues while the plumonary artery blood is well mixed venous blood from the tissue.

The arterio venous difference in the oxygen content represents the amount of oxygen taken up by the tissues for utilization during peripheral circulation per 100 c.c. of blood. Normally the arterio venous oxygen difference falls in the range of 4.0 to 5.5 vol %.

During cardiac decompensation in low output failure the arterio venous oxygen difference averages higher than 6 vol % but there is considerable overlaping of it between a normal and a deranged haemodynamic system. This greater arterio venous difference of oxygen per unit of blood results from the diminished blood flow. In high output failure the arterio venous oxygen difference is proportionally small. Therefore the arterio venous difference would vary inversely as the amount of blood flow and directly as the degree of decompensation.

5 Cardiac output, Cardiac index

The cardiac output is the amount of blood flowing per minute out of the heart Because the cardiac output varies with the oxygen consumption and therefore the surface area it is expressed as the cardiac index or the cardiac output per minute per sq meter of the body surface area The normal resting cardiac index is 3 2 to 3 31 litres per minute in audlts and slightly [1] [4] [23] higher in children The cardiac index is diminished in an upright posture in prolonged standing [28] and by holding breath in inspiration as in valsalva because of diminished venous return However the cardiac output can go as high as 36 litres per minute in athlets during competition

Nature has provided several mechanisms to maintain the cardiac output under physiological and pathological stress and strain on the myocardium such as under exercise and congestive heart failure The following factors can be considered as the compensatory factors in order to maintain an adequate output

1 Heart rate The cardiac output is the product of stroke volume and heart rate In a normal person the heart rate increases to a certain extent under any physiological stress In a decompensated myocardium the heart rate increases disproportionately with any additional stress Thus up to a certain limit increase in heart rate increases the cardiac output

2 Stroke output The stroke output increases to a certain extent until all the cardiac reserve is finished An attempt then is directed towards the hypertrophy of the ventricular musculature to raise the stroke volume Normal heart under exercise will raise both the stroke volume and the heart rate but the failing heart can rarely raise the stroke volume under exercise and hence the minute output has got to be raised mostly by raising the heart rate out of all proportions to the stroke volume In congestive cardiac failure slightest exertion will produce considerable degree of tachycardia without any significant rise in the stroke volume because after a certain point in the development of tachycardia there is not diastolic filling enough to fill the ventricles to accomplish any significant increase in a stroke volume

3 Raising the blood volume When the maintainence of an adequate output fails with the simple compensatory mechanisms like increased heart rate stroke volume and cardiac enlargement then the restoration of the cardiac output may occur by increase in blood volume The progressive increase in the circulating blood volume is a fundamental homeostatic compensatory mechanism

However this increased blood volume itself results into symptoms which we call congestive cardiac failure Thus up to an optimum Physiologic limit increased blood volume produces a higher venous return resulting in an increased ventricular diastolic volume and subsequently in an augmented cardiac output [47] However beyond this optimum limit increase in blood volume *is not able to produce greater cardiac filling nor greater cardiac* output because there occurs a pronounced rise in pressure within the cardiac chambers This leads to the decrease in veno atrial pressure difference which is the effective filling pressure [21] *This happens in later stages of congestive failure when the* cardiac output is considerably diminished

4 Redistribution of blood flow Whenever the cardiac output falls rapidly or *severely arteriolar vasoconstriction* is an important mechanism Arteriolar vasoconstriction of the splanchnic vessels and the extremities spares a greater share of the output proportionally for the vital organs such as brain and the heart [13, 1] The diminished renal flow and therefore the retention of sodium and water due initially to a low cardiac output has been of particular interest as it increases the blood volume and the venous return Although this may transiently augment the cardiac output it ultimately increases the strain and congestion and leads to further increase in failure

As has been mentioned before during the early stages of cardiac failure the resting cardiac index may be in normal range [12, 47] though *there is initially considerable overlap of values As a rule in our experience* coinciding with that of others [1] patients having a cardiac index below 2 5 will be more or less incapacitated The degree of symptomatology however will vary both with the patients personality and other mechanism of physiologic protection such as an elevation of pulmonary arteriolar resistence etc etc In advanced failure the cardiac index can go quite low in our experience down to 1 0 The patients in cardiac failure during exercise [2, 1] cannot increase their cardiac output in proportion to the body s increased needs In other words in a congestive cardiac if the O_2 consumption rises say by 100% the cardiac index may rise by 10 or 15% only and the arterio venous oxygen difference is not able to make up the deficit The failing heart therefore even if it is able to keep the patient symptomless at rest is unable in its adaptability to cope with the additional stress [44] In a normal circulation for every 1 c c rise in oxygen consumption cardiac output is raised by 6 c c but in a failing heart it may be about 3 c c or less for every 1 c c rise in oxygen consumption This is a constant observation of a disproportionate rise between the oxygen consumption and minute output

The cardiac index when extremely low does indicate the severity of the disease However it does not bear any constant relationship with the symptoms or the physical signs [33, 38, 52] or the pathological changes in the myocardium or the pulmonary arterioles or capillaries because most of the symptoms of the congestive failure are caused to a great extent by the compensatory mechanisms rather than by the reduction in the minute output per se

6 Methods of determining the cardiac output

- I a Based on Ficks principle involving gaseous analysis
  - b Based on Ficks principle without gaseous analysis
- II a Dilution method Gas rebreathing Ethyl iodide method
  - b Dye dilutions and the dilution of radio active substances
- III a Roentgenokymography
  - b Pulse pressures and the velocity method
  - c Ballistocardiography

I a Methods based on Ficks principle involving gaseous analysis Out of all methods this is perhaps the most accurate and commonly practised [16] It depends upon the oxygen exchange between blood and alveolar air in the lungs The cardiac output is calculated by determining the oxygen (or any other gas given to breathe) content of mixed venous blood drawn through the catheter from the pulmonary artery the peripheral arterial blood drawn simultaneously through an arterial needle and the minute pulmonary gas exchange at the same time When the minute volume of pulmonary gas exchange (oxygen consumption) is divided by the arterio venous difference of this gas in the blood the result is the number of c c of blood which took part in the delivery of the gas per minute

This is the method practised conventionally at all cardiac centres during the study by cardiac catheterization

b Method based on Ficks principle without gaseous analysis [11] The kidneys are used here as the basis for the exchange of a material sodium para aminohippurate (PAH) or its acetylated derivative paraacetylaminohipuric acid (PACA) is used as the test material mainly because it has a renal extraction of about 90% A dobule lumen catheter is placed with the distal tip about 1 inch beyond the pulmonic valve in the pulmonary artery Through this lumen a

constant amount of PAH or PACA is injected by a drip method (through an infusion pump) into the pulmonary artery and after equilibration mixed venous samples are collected from the proximal lumen in the right ventricle Arterial blood is drawn from the brachial or the femoral artery for determining the amount of PAH or PACA The mixed venous blood from the right ventricle is corrected for its changing plasma level Simultaneous arterial and mixed venous blood contents can be determined Hence the cardiac output can be calculated by the Fick s principle

The other methods are only briefly mentioned below —

II a The patient is breathing and rebreathing the same foreign gas like nitrous oxide acetylene etc [31] to secure equilibrium with the mixed venous blood

b Dye dilution and the dilution of the radio active substances are also used for the purpose This method is useful only in a limited number of cases Besides valid quantitative conclusions can be drawn only after repeated determinations [20]

III a Roentgenokymography

The stroke output is calculated by studying the volume changes of the heart by means of roentgenokymography This method has been full of many errors [7]

b *Pulse pressure and velocity method*

This has been of dubious reliability

c Ballistocardiography

This is very unreliable particularly in heart diseases and during inconstant heart rate [8] [41]

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CHAPTER VII

CALCULATIONS

Vascular resistances

This is analogus to the resistance in the electrical circuit and it is conventionally estimated by Poiseulles law[11] which is based on rigid tube system and continuous flow As the cardiovascular system has a pulsatile flow and elastic walls the data obtained from such equations may not be very exact

In any given circuit of fluid

$$Resitance = \frac{\text{Pressure gradient}}{\text{Volume of flow per unit time}}$$

In order to standardise this way of expression it may be expressed in C G S system (absolute units of fundamental units of force)

Resistance is equal to

$$\frac{\text{Press grad in mm Hg x 98 117 (force of gravity) x 13 59 (sp gr of Hg)}}{\text{Blood flow in c c per second}}$$

Now the pressure in mm of Hg must be converted into dynes/cm² and flow expressed in litres per minute must be converted into cm³/second Thus the equation becomes

$$\text{Resistance} = \frac{\text{Press grad in mm Hg}}{\text{Cardiac output}} \times \frac{13\,59 \times 98\,117 \text{ dynes/cm}^2}{1000 \text{ cm}^3/60 \text{ seconds}}$$

$$\text{or} = \frac{\text{Press gradient}}{\text{Cardiac output}} \times 60 \times 1333\,4 \text{ dynes/second cm}^{-5}$$

Applying this formulla (first used by Aperia 1940)[1] to

1 Pulmonary arteriolar resistance =

$$\frac{\text{Pum art mean} - \text{Pulm vein mean}}{\text{Cardiac output}} \times 60 \times 1333\,4$$

$$\text{or} = \frac{\text{Pulm art mean} - \text{Pulm arterial wedge mean}}{\text{Cardiac output}} \times 60 \times 1333\,4$$

Normal average by various laboratories differs from 70 to 120 In order to be on the safer side values above 200 only should be considered pathological The upper limit of normal accepted in the present study is 150 The pulmonary arteriolar resistance

calculated as shown above certainly indicates the condition of the pulmonary capillaries at the time of the study. Any histological or physiological changes leading to the narrowing of the pulmonary capillary bed is immediately reflected through the resistance. The pulmonary arteriolar resistances in normal individuals have almost a linear correlation with the pulmonary artery diastolic pressures, pulmonary artery systolic pressures and still better correlation with the pulmonary artery mean pressures. In normal individual the increase in the pulmonary arteriolar resistance due to any cause is associated with the elevation of the pulmonary artery systolic, pulmonary artery diastolic and the pulmonary artery mean pressures proportionally.

2. Total pulmonary resistance (including the resistance beyond pulmonary capillaries)

$$= \frac{\text{Pulmonary artery mean} - 0}{\text{cardiac output}} \times 60 \times 1333.4$$

The upper limit of normal taken during the present study is 250.

3. Mitral resistance (including the resistance offered by the left ventricle during diastole)

$$= \frac{\text{Pulm. art. wedge mean}}{\text{cardiac output}} \times 60 \times 1333.4$$

4. Mitral valve restistance (excluding the resistance offered by the left ventricle during diastole)

$$= \frac{\text{Pulm. art. wedge mean} - 5}{\text{cardiac output}} \times 60 \times 1333.4$$

(5 is the assumed left ventricular diastolic pressure)

The validity of this formula is based on the assumption that the left ventricular diastolic mean pressure is 5 mm Hg, which cannot be too far from correct. However if left sided pressures are measured, the resistance can be calculated in more certain terms.

5. Systematic resistance (including resistance offered by the right ventricle during diastole)

$$= \frac{\text{Brach. art. mean} - 0}{\text{cardiac output}} \times 60 \times 1333.4$$

Accepted limits of normal range is from 700 to 1900 units.**

Similarly the resistance of the tricuspid valve in tricuspid stenosis and pulmonic valve in pulmonic stenosis can be calcu-

lated by proper substitution of the data in the formula for resistance of the mitral valve

Right ventricular pressure work. Left ventricular pressure work.

The total energy produced by the heart is split into two parts namely heat and mechanical energy. There is no way of measuring the heat energy. The mechanical energy therefore is the only calculable part of the total work performed by the ventricles. This mechanical energy is spent in two directions —

i. In ejecting a definite volume of blood into the aorta or the pulmonary artery against pressure.

ii. In giving the momentum to that volume of blood which is ejected.

The present study is concerned with the work done by each ventricle in ejecting a volume of blood only. This is only a pressure work which is a fraction of the total work performed.

Work done =

$$\left\{\begin{array}{c}\text{volume of systolic}\\ \text{ejection}\end{array}\right\} \times \left\{\begin{array}{l}\text{Mean elevation of pressure}\\ \text{accompanied by that parti-}\\ \text{cular ventricle}\end{array}\right\}$$

In order to express it in a C.G.S. system and in a standard way for comparative purposes the following formulas are *generally accepted* —

Right ventricular pressure work =

$$\frac{\left\{\begin{array}{c}\text{cardiac index}\\ \times\\ 1.055\\ \text{sp. gr. of blood}\end{array}\right\} \times \left\{\begin{array}{l}\text{Pulm. art. mean}\\ \text{— Right atrial}\\ \text{mean i.e. pressure}\\ \text{elevation by R.V.}\end{array}\right\} \times 13.59\ (\text{sp. gr. of Hg})}{1000}$$

K.gm. per minute per sq. meter

With a normal range of pulmonary pressures the right ventricle generally does not have to work beyond 0.5 to 0.6 K.gm. per min. per sq. meter.

Similarly left ventricular pressure work =

$$\frac{\left\{\begin{array}{c}\text{cardiac index}\\ \times\\ 1.055\end{array}\right\} \times \left\{\begin{array}{c}\text{Brach. art. mean}\\ -\\ 5\ (\text{L. V. dias. pres.})\end{array}\right\} \times 13.59}{1000}$$

Within a normal range of systematic pressures the left ventricular pressure work does not usually rise more than 4 to 5 times the right ventricular pressure work.

Diastolic filling period Systolic ejection period

The exact estimation of the diastolic filling period and the systolic ejection period of the left ventricle can only be obtained from the simultaneous pressure tracings from the left atrium left ventricle and the aorta It is not however considered practical to obtain these tracings in routine cardiac catheterization Gorlin et al suggested that the brachial artery tracing if used for calculating the diastolic filling period or the systolic ejection period gives values very close to those of Lombard cope method * and is believed that brachial artery tracing gives sufficiently consistent and valid results for the purposes of this measurement The diastolic filling period of the left ventricle can be measured from the beginning of the dicrotic notch to the beginning of the upstroke of the subsequent pulse The diastolic filling period calculated this way however includes the isometric contraction and relaxation but in mitral stenosis the atrio ventricular flow probably occurs during a part of these phase and therefore these errors are minimized in mitral stenosis

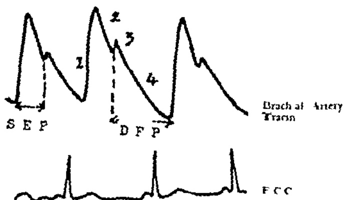


Fig 1

Simultaneous brachial artery tracing and the E C G

1 Ascending limb
2 Pre dicrotic segment
3 Dicrotic notch
4 Post-dicrotic segment
D F P Diastolic Filling Period
S E P Systolic Ejection Period

The systolic ejection period of the left ventricle is measured from the beginning of the upstroke to the beginning of the dicrotic notch of the same pressure pulse However systolic ejection period calculated this way excludes the period of isometric contraction and relaxation although in gross mitral insufficiency the regurgitation probably begins during a part of isometric contraction and continues partly during isometric relaxation Therefore the values obtained for systolic ejection period is little smaller than the actual The regurgitation in mitral insufficiency can occur only when the atrio ventricular pressure gradient of diastole

is reversed in systole This reversal of gradient occurs only late in isometric contraction period and continues only in the beginning of the isometric relaxation period because of the elevated left atrial pressure and the time lag occuring in the reversal of flow Therefore the errors are minimized in mitral insufficiency

The effect of exercise or tachycardia on the diastolic filling period or the systolic ejection period has been shown in the discussion on the normal arterial tracing

Mitral valve stenotic area

From the two accepted formulae in hydraulics Gorlin & Gorlin[1] first derived a formula for the calculation of the mitral valve stenotic area In a system of two chambers communicating by a fixed orifice

1 Flow = (Cc x Area x Velocity

where Cc is the coefficient of contraction

2 Velocity^2 = Cv^2 x 2gh

where g is the force of gravity
h is the pressure gradient
Cv is the coefficient of velocity
(as only a part of pressure is converted into velocity)

or Velocity = Cv x $\sqrt{2gh}$

Solving these two equations for

$$\text{Area} = \frac{\text{Flow}}{Cc \times Cv \times \sqrt{2gh}}$$

(Cc x Cv) value has been derived by Gorlin and Gorlin[1] by comparing the calculated valve areas to actual measured areas at autopsy (cc x Cv) value is different for different valves Because of our limited knowledge of the circulatory haemodynamics this value only supposedly corrects for several practical limitations in the methods This correction factor also includes the pressure conversion factor from mms of Hg to cms of water In case of mitral stenosis the formula has been derived as follows —

$$\textit{Area} = \frac{\text{Flow}}{C \times \sqrt{2 \; 980} \; \sqrt{P_1 - P_2}}$$

$$= \frac{\text{Flow}}{C \times 44.5 \sqrt{P_1 - P_2}}$$

$$= \frac{\text{Mitral valvular flow}}{C \; 44.5 \sqrt{PWm - 5}}$$

5 is the assumed left ventricular diastolic pressure Pulmonary arterial wedge pressure (PWm) is assumed equal to the left atrial mean which is the driving pressure head across the mitral valve and the mitral valvular flow is the rate of flow across the mitral valve i e flow per diastolic second which is equal to

$$\frac{\text{Cardiac Output}}{\text{Diastolic filling period in seconds}}$$

Substituting the value of C as has been worked out by Gorlin & Gorlin [1]

$$\text{Mitral Valve Stenotic Area} = \frac{\text{Mitral valvular flow}}{0.7 \times 44.5 \times \sqrt{Pwm - 5}}$$

$$= \frac{\text{Cardiac output/ Dias filling period}}{31 \times \sqrt{PWm - 5}}$$

$$= \frac{\text{Cardiac output}}{31 \times \text{Dias Filling period} \times \sqrt{PWm - 5}}$$

If the gradient (PWm — 5) is too small formula is likely to give erroneous results In these cases exercise will raise the gradient (PWm — 5) Validity of this formula has been questioned by some people in the field but in our experience it has been of value The applicability of this formula exists only in the presence of pure mitral stenosis without any regurgitation

Mitral valve regurgitant area —

Mitral regurgitation occurs during the systolic ejection period of the left ventricle By the same formula as in mitral valve stenotic area

$$\text{Area} = \frac{\text{Flow}}{Cc \; Cv \; \sqrt{2gh}}$$

$$= \frac{\text{Rate of regurgitant flow during ventricular systole}}{C \; 44.5 \sqrt{h_1 - h_2}}$$

$$= \frac{\text{Rate of regurgitant flow per systolic second}}{C \; 44.5 \sqrt{L \, V \text{ sys mean} - L \, A \text{ Mean}}}$$

$$= \frac{\text{Regurgitant flow/ Systolic ejection period}}{C \; 44.5 \sqrt{L \, V \text{ sys mean} - L \, A \text{ mean}}}$$

$$= \frac{\text{Regurgitant flow}}{\text{C} \times \text{Sys eje period} \times 44.5 \times \sqrt{\text{L V Systolic mean} - \text{L A mean}}}$$

$$= \frac{\text{Total regurgitant flow}}{\text{C} \times \text{S E P} \times 44.5 \times \sqrt{\text{Brach art sys mean} - \text{L A mean}}}$$

The value of C has been calculated by Gorlin & Dexter mostly on hypothetical considerations and approximate value suggested by them is 0.8

Now

Regurgitant flow = Total left vent inflow — systemic output
= Total mitral valve flow — cardiac output

Substituting the value of mitral valve flow from the formula for mitral valve stenotic area

$$\text{Total mit val flow} = \text{M V Ste area} \times \text{Dias fill period} \times 31 \times \sqrt{\text{PWm} - 5}$$

We get

$$\text{M V Regurg Area} = \frac{\text{Total M V flow} - \text{cardiac output}}{0.8 \times 44.5 \times \text{S E P} \times \sqrt{\text{Brach art sys mean} - \text{L A mean}}}$$

$$= \frac{\text{M V sten area} \times \text{D F P} \times 31 \times \sqrt{\text{PWm} - 5}) - \text{card output}}{36 \times \text{S E P} \times \sqrt{\text{Br art sys mean} - \text{PWm}}}$$

There is no way of estimating the mitral valve stenotic area in the presence of regurgitation and therefore there is no direct practical application of this formula

No attempt was made in cases studied at this laboratory to estimate quantitatively the degree of regurgitation from this formula However it is claimed that the ratio $\frac{\text{Regurgitant area}}{\text{Stenotic area}}$ can be estimated and can be used for the fair evaluation of the results of mitral commisurotomy Improvement in function occurs due to the increase in the mitral valve stenotic area and decrease in the mitral valve regurgitant area In either case the ratio $\frac{\text{Mit Val Reg Area}}{\text{Mit Val Ste Area}}$ will diminish

This formula although interesting invites much criticism mainly because it involves many presumed and assumed indirect measurements and values

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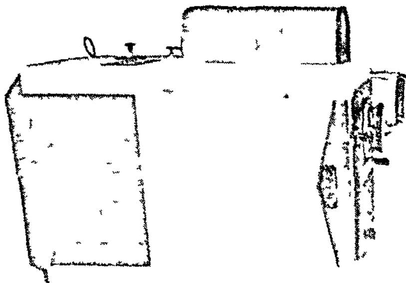
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Part II

CHAPTER 1

VALUE OF CARDIAC CATHETERIZATION IN STUDYING THE DYNAMICS OF MITRAL STENOSIS

The advent of cardiac surgery has been largely responsible for the increased enthusiasm in the study of the mitral valve lesions during the last few years As a result the derangement of haemodynamics in mitral valve lesions is better understood to day than at any time in the past The history of mitral valve disease is still in the making and every day progress is being made Nevertheless certain points have now been established by the clinician at the bed side the surgeon at the operating *table and the pathologist at autopsy*

Although cardiac catheterization studies are not absolutely necessary in every case of mitral disease only such studies can supply the following information which is critically required in the pre surgical evaluation —

i Mitral valve stenotic area in pure mitral stenosis where there is discrepancy between the severity of symptoms and the clinically suspected degree of mitral stenosis

ii Predominance of one mitral lesion over the other in double mitral lesions

iii In pure mitral stenosis the predominant role of mechanical or myocardial factors

iv Detection of associated lesions such as tricuspid or aortic lesions

v Distinguishing mitral valve lesions from primary pulmonary hypertension pericardial effusion etc

The study of mitral valve lesions by catheterization of the right side of the heart does not provide any direct information about the anatomical state of the mitral valve but only the state of right heart and pulmonary circulation as affected by the diseased mitral valve However the study of right heart circulation has proved to be a very valuable addition to the armamentarium of the cardiologist for the evaluation of the mitral disease

If a *definite* diagnosis of mitral stenosis can be made on the basis of clinical findings and usual investigations the right heart catheterization then provides a measure of severity of the stenosis thus providing infor

mation regarding the relationship between the subjective symptoms of the patient and the degree of stenosis Such information is very valuable to the cardiologist in recommending the surgical treatment of mitral stenosis

In order to determine the predominance of mitral stenosis over mitral insufficiency when both are present pulmonary arterial wedge tracing obtained by right heart catheterization may provide useful information In solving this not an uncommon problem the clinician may have to take the help of left heart catheterization which provides the pressure tracing from the left auricle and the left ventricle

The joint efforts of the physiologist the cardiologist and the surgeon have certainly contributed much to the understanding and evaluation of the extent of mitral heart disease

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CHAPTER II

HAEMODYNAMIC CHANGES IN MITRAL STENOSIS

Gross haemodynamics (Two pressure systems)

Roughly the mitral valve opening has to be reduced considerably from a low normal of 4 cm to 1 2 cm² before the compensatory mechanisms are exhausted

It has been conclusively shown in models that the partial obliteration of the mitral opening will essentially produce two pressure systems —

i High pressure lesser circuit i e increased pressure in the left atrium pulmonary artery and the right ventricle

ii Low pressure greater circuit i e diminished systolic ejection lower arterial pressure and initial transient fall in venous pressure

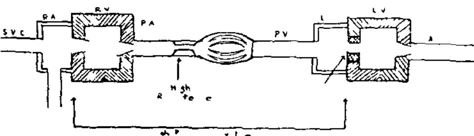


FIGURE 18

Schematic diagram showing the physiodynamics of mitral stenosis. (Two pressure systems)

S V C	Superior Vena Cava	I V C	Inferior Vena Cava
R A	Right Atrium	R V	Right Ventricle
P A	Pulmonary Artery	P C	Pulmonary Capillaries
P V	Pulmonary Veins	L A	Left Atrium
L V	Left Ventricle	M V	Mitral Valve

If as in the above figure the whole system is looked upon as a series of chambers and valves partial obstruction at any point in the system should be met by a proportionate rise in pressure to deal with the degree of obstruction Thus stenosis of the *mitral valve results in a high pressure* area between the mitral valve and the right ventricle As the channel between the mitral valve and the pulmonary valve is entirely unobstructed by any valve action the pressure shift is easily attained all along the system

The high pressure system can be divided into two areas —

i *From the right ventricle to the* secondary branches of the pulmonary artery

ii From the mitral valve to the venous end of the pulmonary capillaries

Although the pressures remain high from the right ventricle to the left atrium the individual chambers maintain their independent characteristics of the pressure patterns Pulmonary artery pressure patterns can be obtained with the tip of the catheter anywhere from the pulmonic valve to the secondary branches of the pulmonary artery From the left atrium to the pulmonary veins the atrial type of the pressure patterns can be obtained The right ventricle has to work much more in order to maintain this high pressure system

Low pressure system

With a tight mitral valve the left ventricle will not be getting enough blood and hence the left ventricular systolic pressure and the stroke output will be reduced As a result the arterial systolic pressure and the pulse pressure will become low

This initial production of two pressure systems is very transient and may even be imperceptible at the onset

In order to increase the flow past the mitral valve in mitral stenosis the following changes take place

i Increased pressure gradient across the mitral valve

ii Increased strength of atrial contraction (unless auricular fibrillation supervenes)

iii Prolonged diastolic filling period of the left ventricle[38]

For a variable length of time depending upon the severity of the stenosis the above mentioned compensatory mechanisms resist the haemodynamic changes in mitral stenosis In the case of auricular fibrillation the compensatory mechanism is greatly handicapped

RIGHT SIDED PRESSURES

Pressures in

i Peripheral veins

Most authors agree that the venous pressure can be normal in a compensated organic heart disease [2] without any sign or symptom of congestive heart failure except perhaps in the instance of the tricuspid disease and constrictive pericarditis

In a chronically decompensated cardiac peripheral venous pressures are high but the pressure gradient between the peri

pheral veins and the right auricle normally seen is frequently reduced to a minimum Increase in this pressure gradient between the venous pressure and the right auricle in a normal heart raises the venous inflow and hence the cardiac output In decompensation however the failing heart is not able to dispose off all the blood that is coming in resulting in an increased diastolic volume and pressure in the ventricle A raised auricular pressure is required to empty against this ventricular residuum

After exercise in a decompensated heart the rise in the venous pressure is sustained for a longer time [3] because the failing heart takes a longer time to deal with the increased venous return

There is no linear relationship between the height of venous pressure and the degree of cardiac decompensation Measurements of the venous pressure therefore cannot be used as a test for cardiac function Only serial determinations can help to judge or prognosticate the efficacy of the therapy

ii Superior and inferior venae cavae

As the catheter is passed from the peripheral vein towards the superior or inferior vena cava the pressure reduces progressively As the catheter comes closer to the right atrium the phasic variations of the auricular type can be seen

iii Right auricle

The pressure in the right auricle is usually raised in the presence of congestive heart failure and this elevation is relatively greater than what occurs in the peripheral veins The gradient between them therefore falls markedly High pressure in the right auricle results mainly because of the elevated right ventricular diastolic pressure [1, 33]

The normal right atrial mean pressure is about 5 mm of Hg in mitral stenosis but if failure is present this pressure is generally found higher than normal i e between 5 and 12 mm of Hg If a tricuspid stenosis is excluded the right atrial mean pressure can be taken for all practical purposes of calculations to represent the driving head of pressure to the right ventricle during diastole The right atrial mean pressure may be elevated or not depending upon the ability of the right ventricle to perform the work load demanded of it

Generally the high right atrial mean pressure is associated with a reduced cardiac output and therefore to that extent the height of the right atrial mean pressure is significant but the converse is not true as patients with a normal right atrial mean pressure can have a diminished cardiac output Hence the right atrial

mean pressure is not indicative of the degree of cardiac decompensation

iv Right ventricular diastolic pressure

In a fully compensated congestive heart failure the right ventricular diastolic pressure is about O In the presence of incompletely compensated right sided failure the right ventricle is not able to discharge all the blood from it and therefore the diastolic *pressure is slightly higher than O As long as the right ventricle* is able to eject a normal stroke output under normal filling pressure it is considered adequate and the right ventricular diastolic pressure the right atrial mean pressure and the peripheral venous pressure remain within normal limits When the right *ventricle becomes inadequate however these pressures begin to* rise

Some or all of the following changes may be observed during the catheterization in the presence of the right sided failure —

a High peripheral venous pressure

b Elevated right atrial mean pressure (to a relatively greater extent than the peripheral venous pressure)

c Diminished pressure gradient between the peripheral veins and the right atrium

d A rise in the mean right ventricular diastolic pressure
This diastolic elevation is often initiated by an early diastolic dip followed by arching up to a plateau like mid and late diastolic rise

e A right ventricular mean systolic pressure rise

f Plumonary hypertension

Right ventricular systolic and mean

There occurs a definite rise in both of upon the total pulmonary re ventricle has to work The *transmitted to the pulmonar* valve The significance of be considered in the disc

PULMONARY HYPERT

By the technique of the righ of the normal and abnormal advanced It has been our symptomatic grade " of a pati tion with pulmonary hyperte correlation has been linear o exertional dyspnoea (class I

pulmonary artery pressure while those who are moderately or markedly debilitated by exertional dyspnoea (class III or IV) may show high degree of pulmonary hypertension

The progress of the mitral stenosis is reflected step by step by the corresponding changes in the pulmonary circulation with secondary strain on the right ventricle Pulmonary artery pressures provide the clinician with more accurate information regarding the functional disability of his patient

It is of great value to observe and correlate the rise in pulmonary artery pressures during physical exercise when the myocardial as well as the pulmonary reserve is called to act upon at their best (see figure 22) The pulmonary artery pressures rise very little after a mild exercise in a normal person [27, 16] but patients with mitral stenosis having mild pulmonary hypertension at rest show markedly abnormal response to moderate exercise [3]

1 Plumonary hypertension and the oxygen consumption

In a normal person the pulmonary artery mean pressure shows practically no rise or only a slight rise (under 10 mm of Hg) until the oxygen consumption rises to 400 c c per minute per sq meter But with excessive exercise raising the oxygen index beyond 400 c c/min/sq meter the pulmonary artery mean pressure may rise significantly The greatest absolute rise recorded by Dexter et al was 16 mm of Hg [1]

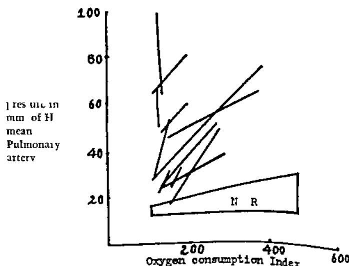


Figure 19

Graph showing the realationship of pulmonary artery mean pressure with the oxygen consumption index

N R Normal Range adapted from Dexter et al J ur ... Phys ... 19..
Cas No ... 3 ... 106 ... 8 109 ... 01 ... 203 (Append...)

In mitral stenosis the pulmonary artery mean pressure at rest may be high and during exercise may elevate inordinately for a small elevation in the oxygen consumption index. By elevating the pulmonary circuit pressures in this way the right ventricle makes an attempt to raise the pulmonary blood flow and eventually the cardiac output.

ii Pulmonary hypertension and the arterio venous oxygen difference

Normally the pulmonary artery mean pressure rises relatively little as compared to the changes in the arterio venous oxygen difference but in mitral stenosis the pulmonary artery mean pressure rises to a disproportionate height in relation to the change in the arterio venous oxygen difference.

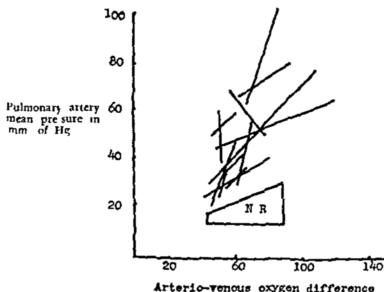


Figure 20
Graph showing the relationship of the arteriovenous oxygen difference with the pulmonary artery mean pressure
N R Normal Range adapted from L Dexter et al Jour Appl Physio 1951
Case No 101 10 1c3 104 105 106 10 108 109 110 201 0 118 (Appendix)

III Pulmonary hypertension and the cardiac index

It can be derived from the figure 21 that unlike normals in mitral stenosis for a small rise in cardiac index there is a striking rise in the pulmonary artery mean pressure.

In mitral stenosis this rise in pulmonary artery mean pressure without a corresponding rise in cardiac index is due to two factors

1 The high head of presure required in the left atrium
2 High pulmonary arteriolar resistance

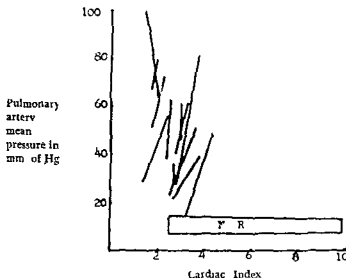


Figure 1

Graph showing the relationship of the cardiac index with the pulmonary artery mean pressure

N R Normal Range adapted from J B Hickman & W H Carmll J C. Invest 1947

Cas No 101 10[illegible] 103 104 [illegible] 106 107 108 109 110 [illegible] 118 Appendix)

In a normal person the ability to raise the pulmonary blood flow and eventually the cardiac output without a corresponding rise in the pulmonary artery mean pressure is due to two factors

1 Opening of new pulmonary capillary channels
2 Dilatation of those capillaries which were already opened

iv Pulmonary hypertension and exercise

Patients with a relatively mild mitral stenosis show pulmonary artery pressure close to normal at rest but during moderate exercise they show abnormally high pressures (2 to 4 times the resting pressure) indicating that their haemodynamic adjustment at rest is maximal without any reserve for extra exertion In those cases where the pressures were recorded during every minute of exercise it was observed that the most common response of the patients with mitral stenosis to exercise was that they raised their pulmonary artery pressure to their greatest elevation by 2 to 4 minutes of exercise

As shown in figure 22 there is a sharp initial rise in pressure which slowly falls after the maximum peak During the post exercise period the fall in pressures may reach below the initial resting level

One is naturally tempted to conclude that one or both of the following two things may be happening during the course of exercise

1 During the initial period of exercise as the cardiac output rises

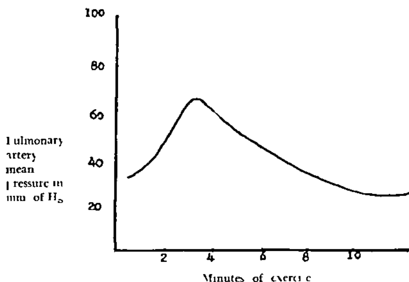


Figure 2

Graph showing the response of the pulmonary artery mean pressure to the exercise in a case of mitral stenosis

the pulmonary arteriolar resistances elevate sharply in order to protect the pulmonary capillaries against the abnormal sudden overload thrown on them resulting in sharp rise of pulmonary artery pressure But after a while there follows a tendency to mutual adjustment between the increased bodily demand for oxygen and the increased amount of blood passing through the pulmonary capillaries by reduction in the pulmonary arteriolar resistances thus permitting diminution in the pulmonary artery pressure The pulmonary artery pressure system adjusts with the cardiac output required under new circumstances at any particular moment

2 Probably the elevated pulmonary artery pressure and the increased pulmonary blood flow may open new channels of alveolar capillaries normally closed

MECHANISM OF PRODUCTION OF THE PULMONARY HYPERTENSION IN MITRAL STENOSIS

The pressures in the pulmonary arterial tree depend upon the following factors one or more of which might be operating at a time —

1 Back pressure rise in the left auricle and pulmonary capillaries due to mechanical obstruction by the mitral valve

2 Pulmonary vasculature and its resistences
   a Dimension and distensibility
   b Resistances due to physiological and pathological changes in arterioles and capillaries

3 Cardiac output
4 Blood viscosity oxygen tension and carbon dioxide content

The relative importance and significance attached to the above factors must be assayed in order to evaluate pulmonary hypertension properly

1 Back pressure

The conventional explanation of pulmonary hypertension in mitral stenosis has been on the mechanical basis The job of maintaining the pressure gradient between the left auricle and the left ventricle during the ventricular diastole across the stenosed mitral valve falls on right ventricle which raises the pressures in the pulmonary circuit enough to meet with the demand despite normal or even reduced cardiac output

During the present study simultaneous pressures taken during mitral commisurotomy in the pulmonary artery and the left auricle have shown that the pressure gradient PAm — LAm is very close to PAm — PWm taken during catheterization (PAm is pulmonary artery mean pressure LAm is the left auricular mean pressure and PWm is the pulmonary arterial wedge mean pressure) In comparing these two pressure gradients of course one has to consider the difference in the physiological states of the patients during catheterization and during surgery under anesthesia

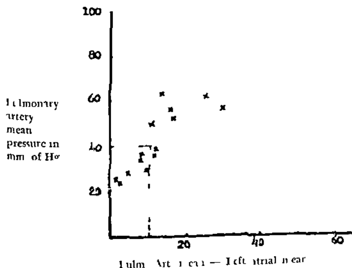


Figure 3

Graph showing the relationship of the difference between the pulmonary artery mean pressure and left atrial mean pressure with the pulmonary artery mean pressure in mitral stenosis

Case Nos 102 103 104 105 106 107 108 110 111 112 113 114 115 116 117 118 (Appendix)

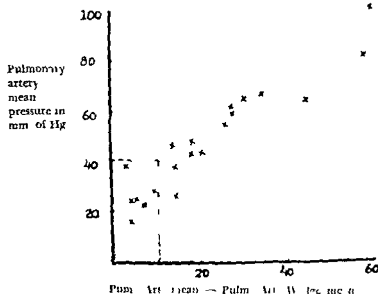


Figure 24

Graph showing the relationship of the difference between the pulmonary artery mean pressure and pulmonary artery wedge mean pressure with the pulmonary artery mean pressure in mitral stenosis

Case No 102 103 103F 104 104E 106 106E 107 107E 108 108E 109 109E 110 111 111E 112 113 113E 114 118 201 202 203 (Appendix)

The graphs in figures 23 and 24 show the PAm — LAm and PAm — PWm plotted against the PAm It can be seen that in patients having a PAm below 40 mms of Hg the gradients PAm — LAm and PAm — PWm are around 10 but patients having a PAm higher than 40 mm of Hg show proportionately elevated gradients

2 Pulmonary vasculature and its resistences

The pulmonary resistences are elevated in the following conditions

a Acquired heart diseases such as mitral valvular disease left ventricular failure etc due to the mechanical damping and raising the back pressure

b Pulmonary disease such as emphysema fibrosis etc

c Congenital heart diseases such as any left to right shunt and Eisenmenger's complex where probably there is defective pulmonary vasculature

A very important role is played by the pulmonary vascular bed in mitral stenosis It is a highly controversial point as to what extent the

pulmonary hypertension can be attributed to the increased pulmonary arteriolar resistences It is not an infrequent experience during catheterization to see some patients with mitral stenosis stand fairly high degrees of pulmonary hypertension even approaching systemic pressure while exercising without any undue breathlessness at the time of measurement Of course the patients with severe pulmonary hypertension do more often have a history of orthopnoea and paroxysmal pulmonary oedema Nevertheless the fact that these patients can stand pulmonary artery pressures approaching to systemic pressures during catheterization without the production of pulmonary oedema would tend to suggest that there is some intermediate factor working between pulmonary hypertension and the production of pulmonary oedema and that this is the development of pulmonary capillary resistances This has been further substantiated by more commonly found pathological changes in the pulmonary capillaries in a long standing pulmonary hypertension These changes have been discussed previously In the present series the majority of cases had only moderate pulmonary hypertension The study of lung biopsies from the operated cases *did not show the pulmonary vascular changes is frequently* as reported by Larabee It is believed therefore that in moderate pulmonary hypertension the changes in pulmonary arterioles and capillaries are more of physiological nature such as vaso constriction and narrowing of the pulmonary vascular bed

The pressure in the left atrium is required to equalize its outflow with the inflow In accomplishing this whenever the left atrium is working at its capacity level help is extended by the following two factors

i Narrowing of pulmonary vascular bed reducing the right ventricular output This partly contributes to the development of the right ventricular failure

ii Smaller output from the left side will result in diminished venous inflow and therefore decreased right ventricular ejection

Thus after a period of inter adjustment between the degree of mitral stenosis left atrial pressure pulmonary vascular bed and the right ventricular activity the net result is the pulmonary hypertension and the diminished cardiac output

The reflex vaso constriction of pulmonary arterioles and/or organic narrowing of the pulmonary vascular bed can explain the observed dissociation between the pulmonary artery pressures and the expected back pressure gradient transmitted from the left atrium These changes in the pulmonary vasculature throw a chronically increased load on the right ventricle which ultimately fails producing the picture of the right ventricular failure

3 Cardiac output

The third factor which can affect the development of pulmonary hypertension is the right ventricular output. In a mitral stenosis inspite of the dynamic attempt to maintain the right ventricular output it is usually lowered or at the best normal. The development of pulmonary hypertension in mitral stenosis therefore can not be said to have been caused by the cardiac output. On the contrary in cases of mitral stenosis associated with myocardial insufficiency giving rise to low output the pressure in pulmonary artery cannot go as high as can be expected in mitral stenosis with adequate myocardium.

4 Blood viscosity, oxygen tension and its carbon dioxide content

These are not altered enough to contribute to the production of the pulmonary hypertension in mitral stenosis.

Conclusion

It is of paramount importance to realize after the above discussion that there exists a high correlation between the degree of pulmonary hypertension and the functional incapacity of the patient, recognising the fact that there exist a number of exceptions to the rule. Sometime there is significant pulmonary hypertension with relatively less impressive symptoms and signs. This can be explained on the basis of disproportionate *changes occuring in pulmonary vasculature and its resistence.* Rarely one sees mild hypertension associated with history of severe symptoms. This may be explained on the basis of psychological make up of the patient.

However it is concluded from the present study that it is not possible to show a linear working correlation between the pulmonary artery pressure as an isolated factor and the degree of mitral stenosis, extent of pathological changes occuring in pulmonary vasculature or the patient's chances of getting paroxysmal attacks of pulmonary oedema. Also it has been hard to correlate the history of paroxymal dyspnoea to the isolated factor of pulmonary artery pressure at rest although the patients with history of paroxysmal dyspnoea did respond with marked abnormality to exercise. There was found no linear relationship between the degree of this abnor*mal response of these patients and the severity or the frequency of their* paroxysmal dyspnoea.

PULMONARY ARTERIAL WEDGE PRESSURE IN MITRAL STENOSIS

Since the time when Hellems et al[6], Langerlof and Werko[29] first advanced the concept that the pulmonary arterial wedge pressure is a good index of the left atrial pressure, if not exactly equal to it, there has been much corroboratory evidence for the concept[4, 11, 28, 27, 19, 2, 3, 25, 5]. This point has been considered previously. The discussion below is based on the assumption that the pulmonary arterial wedge pressure is

the closest reflection of the left auricular pressure and the two are almost equal

1 Pulmonary arterial wedge pressure at rest

Factors which can necessitate the elevated pulmonary arterial wedge pressure are as follows

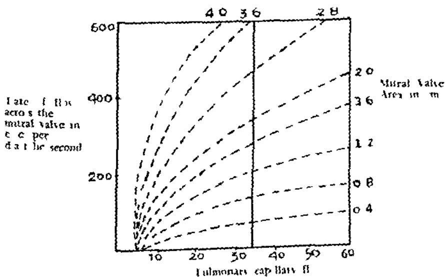
a Degree of mitral stenosis or diminution of mitral valve area
b Cardiac output
c Diastolic filling period
d High left ventricular diastolic pressure

a Degree of mitral stenosis

In order to maintain the required cardiac output PW pressures will have to be elevated depending upon the area of the mitral opening According to Gorlin and Gorlin's hydraulic formula for mitral valve area

PW mean pressure varies as $\frac{\text{Mitral valve flow}}{(\text{Mitral valve area})^2}$

With a smaller valve area PW elevation to raise the mitral valve flow would be tremendously high while with a larger valve area small rise in PW will be able to maintain the necessary mitral valve flow From the present study it is concluded that a person with about 1 cm of mitral valve area may be able to meet with the demand of daily routine of sedentary work without quite exceeding the PW pressure level of pulmonary oedema



Figure

Theoretical curves showing the pressure flow relationship of the Mitral Valve Area
(Adapted from Gorlin et al A H J 1951)

Thus for a certain cardiac output the PW pressures and the mitral valve area are closely related factors The obstruction offered by a small mitral opening causes a compensatory elevation of the PW pressure That which cannot be accomplished by the elevation in PW pressure is suffered by the reduction in cardiac output

Thus high PW pressure is usually a good indication of the severity of a mitral stenosis but normal or slightly elevated PW pressure does not exclude moderately severe mitral stenosis

b Cardiac output

In a mild to moderate mitral stenosis the compensatory mechanism of the myocardium is successful in its attempt to keep up almost normal cardiac output by means of an elevated pressure head in the left atrium or pulmonary capillaries But if the mitral stenosis is moderately severe elevation of the pressure head is not enough to maintain a normal cardiac output The present study confirming the experience of Gorlin R et al [22] showed that with a mitral valve area of 1 cm^2 or less normal cardiac output could not be attained with only one exception

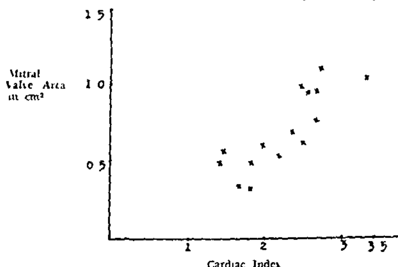


Figure 26

Cardiac Index is in liters/minute/sq meter Mitral Valve Area is in sq cm

Graph showing the relationship between the cardiac index and the calculated Mitral Valve area from Gorlin and Gorlin s formula in pure mitral stenosis

Case Nos 101 102 103 104 105 106 107 108 109 110 111 112 113 114 118 201 202 05 205 (Appendix)

The graph in figure 26 shows the linear relationship of the mitral valve area of less than 1 cm^2 (as calculated from Gorlin & Gorlins formula) [22] with the cardiac index

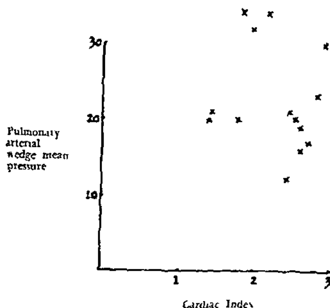


Figure 7

Graph showing the lack of correlation between cardiac index and the pulmonary arterial wedge mean pressure in Mitral Stenosis

Case No (Appendix) 10 104 106 107 108 109 110 111 11 113 114 0 0 0

Figure 27 shows that in order to maintain an adequate cardiac index PW pressures are elevated in all the cases However there is no linear relationship between pulmonary arterial wedge pressure and cardiac index

It should be pointed out here that there are certain conditions like emotional upset or anxiety (as some patients do get in cardiac catheterization room) thyrotoxicosis anemia etc which will record higher or lower PW pressures Thus before a patient *is taken for cardiac catheterization for evalution of mitral stenosis* extrinsic complicating factors should be excluded as far as possible

c Diastolic filling period

In a normal person most of the filling of the left ventricle occurs during early and late diastole of the left ventricle During early diastole there is maximum of blood available in the left atrium to flow into the left ventricle During late diastole atrial systole contributes to the atrio ventricular flow When the diastolic filling period is reduced during the tachycardia of exercise it occurs at the expense of mid diastole so that the total inflow *in the left ventricle does not suffer*

In mitral stenosis the left ventricular filling occurs during the entire diastole at a more or less uniform rate Any reduction in diastolic filling period therefore results in diminution of the total left ventricular inflow In the case of an elevated heart rate the diastolic filling period is smaller and contributes to a reduction in mitral valve flow However if the mitral valve area is only moderately diminished a reduction in the diastolic filling period can possibly be accommodated without raising or only slightly raising the PW pressures

d High left ventricular diastolic pressure

Although the question of high left ventricular diastolic pressure does not arise in pure mitral stenosis it is mentioned here only to discuss its effect on PW pressure independently

Whenever the left ventricle is not able to cope with the work expected of it i e it does not eject as much blood as it receives the left ventricular diastolic pressure is higher and the PW pressure (pulmonary arterial wedge pressure) which is presumably the foreward driving pressure head towards the left ventricle during diastole would have to be raised to the same extent

High left ventricular diastolic pressure is commonly due to the following —

a Active rheumatic myocarditis

b Increased load of work on the left ventricle at rest and during stress

The question of active rheumatic myocarditis is important in the sense that in cases where there is evidence of active myocarditis PW pressure and pulmonary artery pressure should be taken as raised partly due to the left ventricular failure and should not be closely correlated to the degree of mitral stenosis

2 Pulmonary arterial wedge pressure during exercise

In a normal person the pulmonary arterial wedge pressure did not rise significantly within the range of exercise that was performed during the present study

The changes in the pulmonary arterial wedge pressure can also be properly discussed under the following headings —

a Degree of mitral stenosis

b Cardiac output

c Diastolic filling period

d Left ventricular diastolic pressure

a Degree of mitral stenosis

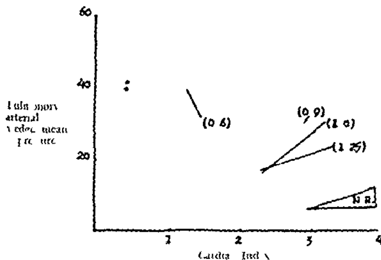


Figure 8

Pulmonary arterial wedge mean pressure in mm of Hg. Cardiac Index is in Liters/Minute/sq. meter. Figures in brackets show Mitral Valve area in cm.

N.R. Normal Range

Graph showing the relationship of the pulmonary arterial wedge mean pressure with the cardiac index during exercise for a particular mitral valve area.

Smaller the valve, higher the pulmonary

Case Nos. 104, 107, 108, 118 (Appendix)

If the required cardiac output is to be established during exercise despite severe mitral stenosis the PW pressure has to rise. In order to accomplish the requisite rise in flow across the mitral valve the PW pressure rise is disproportionately higher with a smaller mitral orifice than with a mitral orifice of the size closer to normal.

As noted previously, cases with mitral valve area of about 1 cm may be able to maintain the cardiac output at a required level without raising the PW pressure beyond the transudation level. These are cases where PW pressures are working at a very narrow margin and therefore any additional demand for cardiac output will easily raise the PW pressure beyond the transudation level.

b Cardiac output

The body requirement for oxygen during exercise is suddenly elevated. With mitral stenosis an attempt is made by the system to meet with the consequent demand for an increased cardiac output by raising the PW pressure. In a moderately severe mitral

stenosis the rise in cardiac output is *less than what would happen* in a normal person In order to make the maximum attempt to meet with the body's demand during effort the PW pressure is elevated depending upon the degree of mitral stenosis In a severe *mitral stenosis PW pressure has to go so high* even during mild exercise to meet with the body demand that it might overshoot the transudation level in the pulmonary capillaries Thus under these circumstances the PW pressure exceeds the plasma osmotic pressure in the pulmonary capillary bed and the signs of the pulmonary congestion begin to appear

c Diastolic filling period

During exercise when the heart rate increases the diastolic *filling period is reduced as is shown in previous chapters* In mitral stenosis where the flow across the mitral valve occurs more or less uniformly during the entire period of diastolic filling any reduction in diastolic filling period will cut down the total mitral valvular flow This factor also has to be compensated by the rise in PW pressure

Thus there are two changes occurring in the haemodynamics of mitral stenosis during exercise which relate to an increase in heart rate —

a Increased body requirement for cardiac output which results in tachycardia

b Restriction in mitral valvular flow due to reduced diastolic filling period that results from the tachycardia

An attempt for compensation can be considered in the following two factors

a Elevated PW pressure which is the driving head across the *mitral valve*

*If this required level of PW pressure is higher than the transudation level in the pulmonary capillaries pulmonary conges*tion or pulmonary oedema may result If the required level in *PW pressure is higher than the pumping ability* of the right ventricle congestive circulatory failure occurs

b Prolongation of the total systolic ejection period of the left ventricle

In mitral stenosis during exercise a prolonged systolic ejection period of the left ventricle is the natural result of a shortened diastolic filling period If the prolongation of the systolic ejection period is due to an associated aortic valvular lesion or

a higher systemic resistance due to any other cause the diastolic filling period is further reduced These associated conditions can make the problem of evaluating the mitral stenosis a very critical one which has to be faced not infrequently in practice

d Left ventricular diastolic pressure

Normally the left ventricle is able to take care of the increased amount of blood flow across the mitral valve during exercise without any significant rise in its diastolic pressure In other words the normal left ventricle is competent enough to eject as much blood as it receives during the strain of exercise

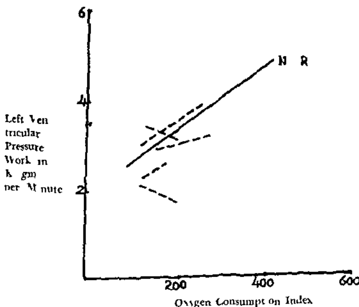


Figure 9

Graph showing the relationship of the left ventricular pressure work with the oxygen consumption index during exercise

N R Normal Range

Case N 10 106 10, 108 118 (App nIx)

As shown in figure 29 the left ventricular pressure work is actually less in mitral stenosis during exercise than in a normal person mainly because of the drop in cardiac index This can be viewed as a protective mechanism that guards against pulmonary flooding However left ventricular pressure work does increase in both normal and mitral stenosis during exercise

If other causes of ventricular failure like active myocarditis high systemic resistance etc are excluded, it appears that left ventricular diastolic pressure should not rise Therefore the left

stenosis the rise in cardiac output is less than what would happen in a normal person. In order to make the maximum attempt to meet with the body's demand during effort the PW pressure is elevated depending upon the degree of mitral stenosis. In a severe mitral stenosis PW pressure has to go so high even during mild exercise to meet with the body demand that it might overshoot the transudation level in the pulmonary capillaries. Thus under these circumstances the PW pressure exceeds the plasma osmotic pressure in the pulmonary capillary bed and the signs of the pulmonary congestion begin to appear.

c *Diastolic filling period*

During exercise when the heart rate increases the diastolic filling period is reduced as is shown in previous chapters. In mitral stenosis where the flow across the mitral valve occurs more or less uniformly during the entire period of diastolic filling any reduction in diastolic filling period will cut down the total mitral valvular flow. This factor also has to be compensated by the rise in PW pressure.

Thus there are two changes occurring in the haemodynamics of mitral stenosis during exercise which relate to an increase in heart rate —

a Increased body requirement for cardiac output which results in tachycardia

b Restriction in mitral valvular flow due to reduced diastolic filling period that results from the tachycardia

An attempt for compensation can be considered in the following two factors

a Elevated PW pressure which is the driving head across the mitral valve

If this required level of PW pressure is higher than the transudation level in the pulmonary capillaries pulmonary congestion or pulmonary oedema may result. If the required level in PW pressure is higher than the pumping ability of the right ventricle congestive circulatory failure occurs.

b Prolongation of the total systolic ejection period of the left ventricle

In mitral stenosis during exercise a prolonged systolic ejection period of the left ventricle is the natural result of a shortened diastolic filling period. If the prolongation of the systolic ejection period is due to an associated aortic valvular lesion or

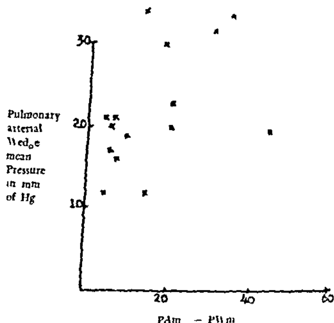


Figure 30

PAm Pulmonary Artery mean pressure

PWm Pulmonary arterial Wedge mean pressure

Graph showing the relationship of the pulmonary arterial wedge mean pressure and the difference between the pulmonary artery mean pressure and the pulmonary arterial wedge means pressure

The figure shows the pattern of Post capillary Pulmonary Hypertension

Case Nos 102 103 104 106 107 108 [illegible] 112 [illegible] 118 201 20 (Appendix

$\frac{PAm - PWm}{PWm}$ therefore is greater than one Hence the pulmonary hypertension is active in origin

It is concluded therefore that whenever the ratio $\frac{PAm - PWm}{PWm}$ greater than one pulmonary vasculature is offering considerable obstruction to the flow in the nature of functional vaso constriction or organic changes in pulmonary arteriolar walls

Figure 31 shows that as the pulmonary artery pressure rises the PWm pressure also rises proportionately till the PWm pressure reaches the level of the osmotic pull of plasma proteins and the gradient between the PAm and PWm changes very slightly But once the PWm pressure reaches close to the pulmonary oedema level (around 35 mm of Hg) or beyond the PWm pressure tends to be fixed However the PAm pressure kept on rising and the gradient therefore rises out of all proportions to the PAm pressure level

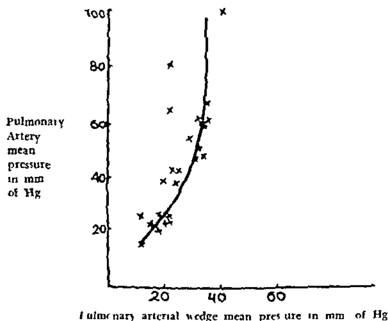


Figure 31

Graph showing the definite relationship of the pulmonary arterial mean pressure with the pulmonary arterial wedge mean pressure in mitral stenosis

Case Nos 103 103E 104 104E 106 106E 107 107E 108 108E 109 110 111 111E 113 114 118 118E 201 202 20 10 (Appendix)

During exercise the PWm pressure does not rise as much as the PAm does (compare figure 32 with 19) particularly because PWm pres

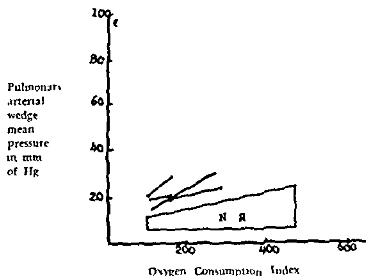


Figure 32

Graph showing the relationship of the oxygen consumption index with the pulmonary arterial wedge mean pressure in mitral stenosis

Case Nos 103 106 107 108 (Appendix)

sure has a tendency to be fixed at the osmotic transudation level in pulmonary capillaries

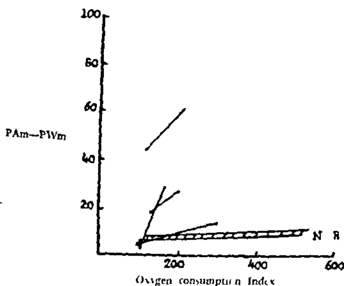


Figure 33

Graph showing the relationship of the oxygen consumption index and the difference between the pulmonary artery mean pressure and the pulmonary arterial wedge mean pressure in Mitral Stenosis

The figure shows a pattern of post capillary pulmonary hypertension

PAm Pulmonary artery mean pressure

PWm Pulmonary arterial wedge mean pressure

Case Nos 103 100 10, 108 (Appendix)

As can be seen in figure 33 PAm — PWm gradient naturally rises more or less like PAm during exercise

OXYGEN CONSUMPTION ARTERIO - VENOUS OXYGEN DIFFERENCE CARDIAC OUTPUT AND THEIR RELATIONSHIP WITH THE PWm PRESSURE

The oxygen consumption arterio venous oxygen difference and the cardiac output are closely inter connected and therefore all the three can be properly discussed together

The normal oxygen index oxygen consumption per minute per sq meter of the body surface area) in adults as accepted during the present study in a resting recumbent state is about 130 c c (normal variation being 100 to 160 c c) Douglas * estimated that the oxygen index at rest increases 1 4 times when standing erect 2 7 times when walking 2 miles per hour and 3 8 times when walking 3 miles per hour All the patients of mitral stenosis in the present study had the oxygen index raised between 2 5 to 4 times that at rest and therefore the degree of exertion taken by these

people can be roughly estimated It may be mentioned here that the air *ventilation* index in these patients varied from 2 4 to 3 5 litres per minute *per sq* meter

A normal person generally has an arterio *venous* oxygen difference of between 4 and 5 5 volumes % which rises slightly during mild to moderate exercise If the oxygen index is increased beyond 400 c c the additional demand is dealt with mostly by rise in the cardiac output and there is hardly any additional rise in the arterio venous oxygen difference Thus the normal system finds it easier to elevate the cardiac *output* than to extract more oxygen from the venous blood For example *when the* oxygen index increases 5 times the arterio venous oxygen difference *may rise only* two times and therefore cardiac output only 2 5 times that at rest

The rise in cardiac output during exercise in the normal is accomplished to a large measure by increasing the heart rate and to a small *extent by* a rise in the stroke volume

In the present *series* all *the* cases of mitral stenosis showed during exercise a smaller rise in *cardiac index* than would normal individuals do In mitral stenosis there are *limitations in* raising the cardiac output Increased tissue oxygen demand therefore *is obtained* by extracting more oxygen from the available volume of blood in *circulation and thereby* elevating the arterio venous oxygen difference The *oxygen index in* all the cases was observed to be within the normal range

However it can be *seen from* figure 34 that although some of the cases have an arterio-venous *oxygen difference* within the normal range at rest they show an abnormal *response during exercise* Thus the arterio venous difference showed a rise *disproportionate to that of the* oxygen consumption index during exercise These abnormally high arterio-venous oxygen differences and the low cardiac output serve as an index *to the* patients inability to adjust fully to the situation of stress

Thus in all *the patients in the* present series the rise in arterio venous oxygen difference *was exaggerated* during exercise and the increase in the cardiac index was *markedly limited* Unlike the normal person the oxygen requirement rose disproportionately *higher* than could be compensated for by a rise in cardiac output The function of increased oxygen supply therefore mainly operated through the increased oxygen extraction from the available circulating blood As a rule for a certain *rise in* oxygen demand due to effort the greater the arterio venous oxygen *difference* the less *efficient* is the system This failure to elevate the cardiac *output in proportion* to the oxygen index is a guide to the limitation of the *patients* adaptability *to* the new situation created during

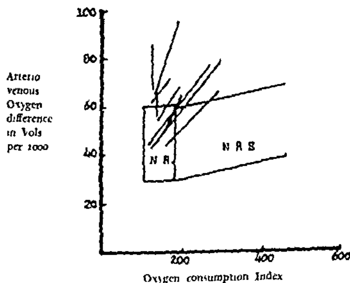


Figure 34

Graph showing the relationship of the oxygen consumption and that of the arterio venous oxygen difference in mitral stenosis at rest and during exercise

The figure shows a disproportionate elevation of arterio venous oxygen difference during exercise

N R Normal range at rest

N R E normal range during exercise

Case Nos 102 103 104 105 106 107 108 109 110 118 (Appendix)

exercise However this restriction in cardiac output did not have any direct relationship with the severity of symptoms the degree of mitral stenosis or the extent of organic pathological changes in the lungs

In one case of severe mitral stenosis the cardiac index actually diminished during exercise (Ref figure 19) A possible explanation that can be offered for this is that the cardiac output rises initially for the first few moments of steady exercise but then drops as the myocardial reserve is exhausted and can no longer cope with the continuing increasing demand In conclusion it was observed that in order to maintain an adequate oxygen index at rest in these patients, the cardiac output and the arterio venous oxygen difference had to adjust in a reciprocal fashion (the former falling and the latter rising)

Relationship with the 'PWm pressure

The inter relationship of the cardic output severity of mitral stenosis and the height of PWm pressure has been considered before In a patient of severe mitral stenosis if the demand for cardiac output is high, the PWm pressure has to be high to maintain an adequate flow across the mitral valve In another patient with a similar degree of mitral stenosis

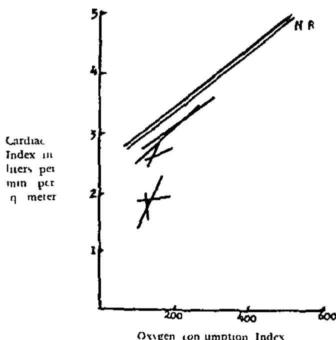


Figure 35

N R Normal range

Graph showing the relationship of the oxygen consumption index with the cardiac index The figure shows a disproportionately smaller rise in cardiac index during exercise Case Nos 103 104 105 106 107 108 109 110 (Appendix)

if the demand for the cardiac output is less even a normal PWm pressure may be able to maintain the necessary flow

The increased oxygen demand *during exercise is of sudden onset* and the system makes an attempt to raise the cardiac output by elevating the PWm pressure to increase the pressure gradient across the valve Of course the patient with very severe mitral stenosis may not be able to raise the cardiac output at all as even at rest he is operating at a maximum reserve adjustment

The limitation in ability to elevate the cardiac output under stress in the patients presented here was considered to be due to the following factors

1 Disharmony between the severity of mitral stenosis and the efficiency of the right ventricle

Despite the hypertrophied right ventricle the rise in pressure in the pulmonary circuit is not adequate to accomplish the necessary *flow across the mitral valve This is considered* to be a factor of prime importance in patients with the long history of the right sided congestive heart failure

ii Voluntary limitation of the willingness on the part of some patients was observed because of severe dyspnoea resulting from the sudden rise in pulmonary pressure particularly when the patient is breathing through a closed circuit for the determination of the cardiac output during effort

iii High pulmonary arteriolar resistances The narrowing of the pulmonary vascular bed which increases in a case of long standing mitral stenosis may be due to pathological changes in pulmonary arteriolar walls (The narrowing developed to protect the pulmonary capillary bed against the flooding from the high pulmonary artery pressure) This is an additional load on the overworking right ventricle

iv In severe mitral stenosis an increase in heart rate during exercise will decrease the diastolic filling period thereby reducing the total left ventricular inflow and the stroke volume

Conclusion

The above four factors tend to restrict the cardiac output while the elevated PWm pressure and the increased heart rate under stress represent the factors counterbalancing the above four factors

VASCULAR RESISTANCE
RIGHT VENTRICULAR PRESSURE WORK
LEFT VENTRICULAR PRESSURE WORK

Pulmonary arteriolar resistance —

$$\text{Pulmonary arteriolar resistance} = \frac{\text{PAm} - \text{PWm}}{\text{Cardiac output}} \times \text{constant}$$

The pulmonary arteriolar resistance varies directly as the pressure gradient PAm — PWm and inversely as the cardiac output

It is important to mention here the relationship between the LAm (Left atrial mean) pressure and the cardiac output With a stenotic mitral valve the LAm pressure having made an initial attempt to raise the cardiac output has a tendency to remain unaffected against further rise by a pre capillary block by pulmonary arteriolar spasm and/or sclerosis The right ventricle at this point is still efficient and raises the pulmonary artery pressure, only a fraction of which is carried through the spastic pulmonary arterioles Elevation of the pulmonary arteriolar resistances is reflected through the disproportionate rise of the pulmonary artery pressure from the PWm pressure

There is little rise seen in the pulmonary arteriolar resistance until the mitral valve area approaches 1 0 to 0 75 cm² but below 0 75 cm² a precipitant rise in the pulmonary arteriolar resistance is seen (Ref figure 36) This rise in pulmonary arteriolar resistance leads to a diminished

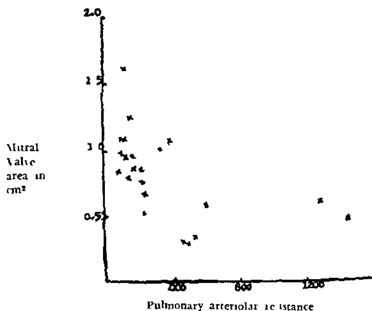


Figure 36

Graph showing the relationship of the pulmonary arteriolar resistance with the mitral valve area in mitral stenosis

Case Nos 102 103 104 105 107 108 109 110 111 112 115 201 203 205 (Appendix)

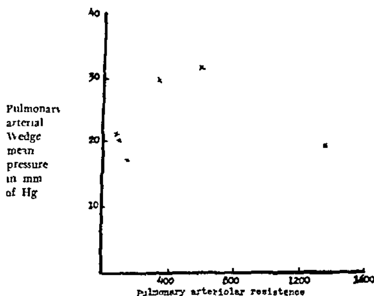


Figure 37

Graph showing the relationship of the pulmonary arteriolar resistance with the pulmonary arterial wedge mean pressure in mitrol stenosis

Case Nos 103 104 106 107 108 109 110 111 112 115 118 201 202 205 (Appendix)

cardiac index This diminution in cardiac index will reduce the pulmonary flow which in turn will prevent the flooding of the pulmonary capillaries

Thus the pulmonary arteriolar resistance behaves as a physiological brake and acts as a governor to limit the right ventricular output inspite of the high pulmonary artery pressure in order to protect the pulmonary capillaries from the pulmonary oedema

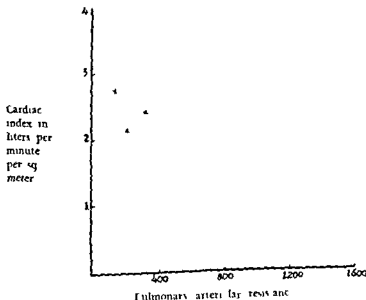


Figure 35

Graph showing the relationship of the pulmonary arteriolar resistance with the cardiac index in Mitral Stenosis
Case Nos 102 103 104 106 107 108 109 [illegible] [illegible] [illegible] [illegible] 114 118 201 202 203 (Appendix)

The elevated pulmonary arteriolar resistance functioning as a protection to the pulmonary capillaries *also further curtails the cardiac output* because the right ventricle has to work against the greater resistance Thus a close interconnection amongst the pressures in the pulmonary circuit pulmonary arteriolar resistance and the cardiac output is well delineated

In a couple of instances elevated PWm pressures rose to oedema level during effort and in this *situation the elevated resistances were inade*quate to counteract the right ventricular efficiency as Hamilton put it like putting your foot on the brake while accelerating with the car in gears"[34]

A severe degree of mitral stenosis of some standing demands constantly *high pressures* in the pulmonary capillaries thereby necessitating the development of high pulmonary arteriolar resistances The evidence of this high pulmonary arteriolar resistance is seen pathologically in the

vascular walls[26, 3] However these elevated pulmonary arteriolar resistances have been observed to be partially reversible following the mitral valvuloplasty[15] *It is believed therefore that these resistances are partly on the basis of physiological vasoconstriction in addition to the pathological* changes in the pulmonary arterioles and capillary walls

Thus the elevated pulmonary arteriolar resistances change the pulmonary circulatory dynamics in two ways —

i *They protect the* pulmonary capillaries *by damping the* pulmonary artery pressure and thereby diminishing the pulmonary *flow* or the cardiac output

ii At the same time high resistances increase the work load of the right ventricle and can exceed the pumping ability of the right ventricle resulting in the right sided *heart failure*

In the course of mitral stenosis a stage comes when the pulmonary arteriolar resistance stays at such a level that the right ventricle is just able to take it and the pulmonary capillary pressure does not rise beyond the pulmonary oedema level This is the stage of relatively less symptoms of pulmonary congestion in the face of progressive disease Soon after this the symptoms of right heart failure starts developing when either the pulmonary arteriolar resistances attempt further elevation or the right ventricle becomes unable to cope with the resistances any longer Further consideration of the role of pulmonary arteriolar resistance in producing the clinical picture will be undertaken in the discussion of the Mechanism of symptoms and signs

During exercise the pulmonary arteriolar resistance was observed to rise considerably With the sudden demand of a rise in the cardiac output both PAm and PWm rise but because of the increased resistance at the pulmonary arteriolar level the rise in PWm is relatively smaller than the rise in PAm and hence the gradient PAm—PWm increases to a greater extent Thus in elevation in the pulmonary arteriolar resistance appeared to be a factor causing a disproportionate rise of the PAm—PWm gradient with the increase in cardiac output during effort Sometimes during effort the cardiac output may even fall through

The cause of the high pulmonary arteriolar resistance in cases of mitral stenosis both at rest and during exercise is still not completely understood The following factors have been considered in the literature as contributory —

i Organic changes in the structure of the pulmonary arteriolar walls as described before

ii Physiologic reflex vaso constriction in the smaller pulmonary arterioles

iii Increased production of nor adrenaline in cases of mitral stenosis the nor adrenaline being selectively active in lungs [10]

iv The changes observed in pleural pressure during effort can produce increased degree of tension in smaller pulmonary vessels

v Autonomic participation Autonomic ganglion blocking agent tetra ethyl ammonium chloride lowers the pulmonary vascular resistance [2]

vi Anoxia Slight anoxia is sometimes observed due to the pulmonary capillary congestion This anoxia may initiate the elevation of the pulmonary vascular resistance [5, 31]

Total pulmonary resistances

$$\text{Total pulmonary resistances} = \frac{\text{PAm in mm of Hg} - \text{O}}{\text{Cardiac output in liters per min}} \times \text{constant}$$

Thus the total pulmonary resistances vary directly as the pulmonary artery mean pressure and inversely as the cardiac output

Cardiac output in liters per minute

6

4

2

1000 2000 3000

Total pulmonary resistance

Figure 39

Graph showing the relationship of the total pulmonary resistance with the cardiac output in mitral stenosis

Case Nos 101 102 103 104 105 106 107 108 109 110 111 112 113 114 201 202 (Appendix)

Inverse relationship was observed between the total pulmonary resistances and the cardiac output

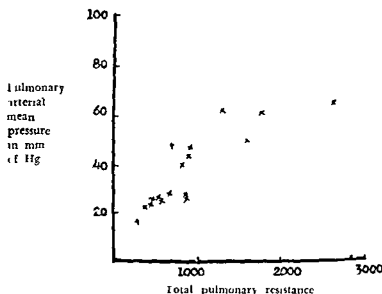


Figure 40

Graph showing the relationship of the total pulmonary resistance with the pulmonary arterial mean pressure in mitral stenosis

Case Nos 101 102 103 104 10 10 1 108 109 110 111 112 113 118 201 202 203 (Appendix)

Significant correlation was observed between the total pulmonary resistance and the pulmonary artery mean pressure

Mitral resistance Mitral resistance = Total pulmonary resistance — pulmonary arteriolar resistance

$$= \frac{\text{PWm}}{\text{cardiac output}} \quad \text{constant}$$

(Mitral resistance includes the resistance offered by the left ventricle during diastole)

Similar relationship of the mitral resistance as the total pulmonary resistance with the cardiac output was observed (Figure 41)

Relationship of the mitral resistance with the PWm is shown in figure 42 As the mitral resistance increases PWm increases proportionally up to the plasma osmotic pressure level

Mitral valve resistance

$$\text{Mitral valve resistance} = \frac{(\text{PWm} - 5) \text{ in mm of Hg}}{\text{Cardiac output in liters/min}} \times \text{constant}$$

(5 is the assumed left ventricular diastolic pressure in the absence of left ventricular failure)

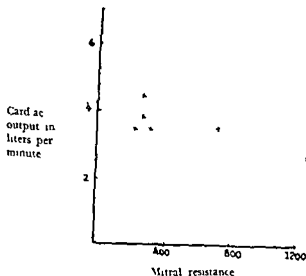


Figure 41

Graph showing the relationship of the mitral resistance with the cardiac output in mitral stenosis

Case Nos 101 102 104 106 107 108 109 110 111 112 113 114 118 201 202 203 (Appendix)

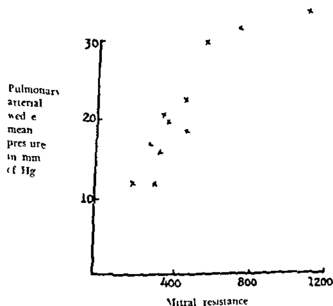


Figure 42

Graph showing the relationship of the mitral resistance with the pulmonary arterial wedge mean pressure in mitral stenosis

Case Nos 102 104 10 108 109 110 111 112 113 118 201 202 (Appendix)

(Mitral valve resistance does not include the resistance offere by the left ventricle during diastole)

No separate graph for the mitral valve resistance is drawn becaus no significant difference is expected from the mitral resistance unle there is elevated left ventricular diastolic pressure as in mitral insufficien or aortic disease or left ventricular failure

Systemic resistance With a severe degree of mitral stenosis the *is a slight drop in systemic pressure and a considerable reduction in th* cardiac output

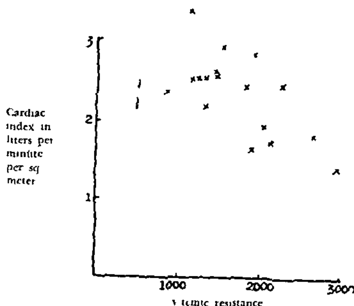


Figure 43

Graph showing the relationship of the systemic resistance and the cardiac index in mitral stenosis
Case Nos 101 102 103 104 105 106 107 108 109 110 111 112 113 201 02 203 203 (Appendix)

Figure 43 shows that with the drop in the cardiac index the systemic resistance increases The correlation seen is quite consistent with the *theoretical expectation The increase in systemic resistance is probably* mainly the result of the reduction in cardiac output and represents a diminution in area of the systemic vascular bed designed primarily to maintain the perfusion pressures to the vital organs

Right Ventricular Pressure Work —

$$\text{Total right vent pres work} = \frac{\text{C I} \times 1.055 \,(\text{PAm} - \text{RAm}) \times 13.6}{1000}$$

kg m /min /sq meter

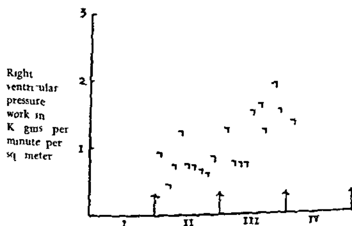
The right ventricular work spent in overcoming the pulmonary arteriolar resistance $= \dfrac{(\text{C I} \times 1\,055)\ (\text{PAm} - \text{RAm} - \text{PWm}) \times 13\,6}{1000}$

The right ventricular work spent in overcoming the mitral resistance $=$

$$\frac{(\text{C I} \times 1\,055)\ (\text{PWm} - \text{RAm}) \times 13\,6}{1000}$$

The part of the right ventricular work is used up overcoming the pulmonary arteriolar resistance and the part in overcoming the resistance beyond the pulmonary arterioles (mitral resistance) Generally the major part of the work is spent in mitral resistance except when the pulmonary arteriolar resistance is exhorbitantly high

Because of the high total pulmonary resistance the right ventricular work is increased despite the reduction in cardiac output Ultimately a stage comes when the right ventricular myocardium at its maximum pumping capacity is not able to cope with the increasing resistances The ensuing right ventricular failure thereby further reduces the cardiac out put The point of the right ventricular failure also depends upon the condition of the myocardium and the presence or absence of active rheumatic myocarditis



Symptomatic grades according to the criteria of the New York Heart Association

Figure 41

Graph showing the right ventricular pressure work in 4 different symptomatic grades according to the criteria committee of New York Heart Association

Case No 101 10 103 104 10 106 10 108 109 110 111 11 115 114 118 01 20 0 20 (Appendix)

In the present study a functional grading of the cases of mitral stenosis [14] was done Out of 9 cases in grade II only one showed the right

ventricular work higher than 1 Kg m/min/sq meter Out of 10 cases in grade III and IV only 3 showed their right ventricular work lower than 1 (Figure 44)

When the myocardium is working at a diastolic fibre length greater than the maximum reserve, efficiency declines An increase in demand on the right ventricle at this time is reflected in an elevated right ventricular diastolic pressure due to residual diastolic blood not emptied by the failing chamber The right ventricle is able to take up quite a high work load even after the onset of failure, as can be seen from the cases of grade III & IV, but it does so with the decreasing efficiency

During exercise the right ventricular work increases very sharply In patients with high pulmonary resistances at rest the rise in the right ventricular pressure work is very sharp

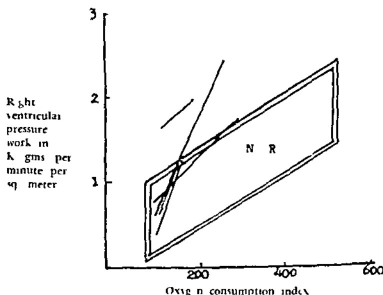


Figure 45

Graph showing the increase in the right ventricular pressure work with the rise of oxygen consumption index during exercise in mitral stenosis

N R Normal Range adapted from Corlin et al A H Jour 1951

The elevated pulmonary resistances demand much higher work from the right ventricle on the slightest effort in order to accomplish the adequate rise in the cardiac output Thus the right ventricle which is working on the verge of failure can be thrown out of gears with minimum effort These demands made from a chamber already stressed to its maximum capacity is the main contributory factor in producing the right ventricular decompensation in cases of mitral stenosis

Left Ventricular Pressure Work —

$$\text{Left ventricular work} = \frac{(C\ I \times 1\ 055)\ (BAm\ \text{or}\ FAm - 5) \times 13\ 6}{1000}$$

Kg m /Min /sq meter

(BAm is Brachial Artery mean pressure)
(FAm is Femoral Artery mean pressure)

In a case of mitral stenosis the total left ventricular inflow is small and therefore the left ventricle has to eject a smaller amount of blood Thus with a smaller cardiac output left ventricle become a relatively relaxed chamber Although the systemic resistance may be little higher the left ventricular pressure work is slightly lower in mitral stenosis at rest than in the normal

During exercise the left ventricular work changes are roughly parallel to the changes in the cardiac index (Refer figures 28 and 35)

DIASTOLIC FILLING PERIOD
MITRAL VALVE STENOTIC AREA

Diastolic filling period —

The magnitude of the diastolic filling period was observed not to follow any definite rules Some times severe degree of mitral stenosis is associated with shorter diastolic filling period than in moderate degree of mitral stenosis and vice versa

During exercise when there is a sudden rise in body demand to increase the cardiac output the diastolic filling period diminishes Any condition leading to tachycardia curtails the diastolic filling period to that extent It has been mentioned previously that unlike normals any reduction in diastolic filling period in mitral stenosis will diminish considerably the mitral valvular flow

Mitral valve stenotic area —

Questions have been raised regarding the validity of Gorlin and Gorlin s hydraulic formula [3] for the calculation of the mitral valve stenotic area Nevertheless during the present study it was believed that with an ideal technique and in cases of pure mitral stenosis the formula has a logical application and one is not likely to be too far from the actual size of the valve In our experience a close check was observed between the calculated area and that estimated by the surgeon s finger However it was very well realized that the mitral valve orifice being irregular it is hard to get any exact estimate of the mitral valve area by the surgeon s finger This is true even at autopsy Thus both the ways the method of

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calculating the mitral valve area by the formula and the method of measuring the mitral valve area by the surgeon's finger are subject to some inaccuracy and therefore close agreement was not expected

The haemodynamic implications of different magnitudes of the mitral valve area have been discussed previously The mitral valve area, the mitral valve flow and the PW' pressure are very closely connected and the immediate repercussion of one on the other have been considered fully in the discussion of the PW

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CHAPTER III

HAEMODYNAMIC CHANGES IN PREDOMINANT MITRAL REGURGITATION

(Double mitral lesions)

Gross Haemodynamics in Predominant Mitral Regurgitation

Predominant mitral regurgitation with mitral stenosis The present study concerns the double mitral lesions Possible changes in the dynamics of mitral insufficiency in the presence of mitral stenosis is discussed

A severe degree of mitral stenosis cannot exist with a severe degree of mitral insufficiency and vice versa Now a days with more and more opportunities available to study the mitral valve by digital palpation it is realised that there is a certain amount of inverse relationship observed in the existence of mitral stenosis and the mitral insufficiency One is the complement of the other [1]

The cases presented here as of predominant mitral insufficiency were diagnosed on the basis of the clinical findings electrocardiography cardiac fluoroscopy and finally by the height of the v wave in the pulmonary arterial wedge tracing or the left atrial tracing All the cases were presented in the cardio-vascular surgical conference of the Montefiore hospital for proper evaluation In some of these mitral regurgitation was confirmed by the surgeons feeling the regurgitant jet at valvotomy No one particular criterion before valvotomy was taken as a sole guide but the over all picture was taken into consideration to decide the degree of mitral regurgitation

Changes in the arterial tree Both mitral stenosis and mitral insufficiency if of sufficient degree will tend to reduce the left ventricular aortic ejection In the patients observed the cardiac output was reduced but the systolic and the diastolic pressures in the arterial tree were not proportionally reduced Therefore peripheral vasco-constriction was assumed

Changes in the left atrium and the left ventricle The reflux of blood in the left atrium during the ventricular systole increases left atrial volume and the pressure in the left atrium rises during ventricular systole The elevated driving head of pressure in the left atrium contributes to better left ventricular filling during the ventricular diastole In a mild insufficiency the left ventricle with a greater diastolic size expels larger

total volume of blood part of which again regurgitates through the mitral valve The share of aortic discharge however comes close to normal In a moderate to severe degree of mitral insufficiency, the aortic discharge is below normal and the cardiac output therefore drops

Stabilised conditions are established as the progress of the lesions is stopped The left atrial pressure and the cardiac output become fixed at a certain level The systemic output is equal to right ventricular output

The maximum regurgitation occurs during the systolic ejection period of the left ventricle Very little of it occurs during the isometric contraction phase proto diastole and perhaps the beginning of the isometric relaxation The duration of the left ventricular systole in mitral insufficiency is similar to that in normals[3]

Changes in the pulmonary circulation The increased volume of blood proximal to the mitral valve is accommodated mainly in the enlarged left atrium pulmonary veins and also in the newly opened pulmonary capillaries

Change in pressures in the right atrium and the right ventricle The right atrial venous pressure does not change due to the regurgitation as a rule The patients with mild to moderate mitral insufficiency observed in our series did not have right atrial or right ventricular pressures or right ventricular work values that were significantly different from the simultaneously observed group of patients with pure mitral stenosis

Conclusion The mitral regurgitation induces in the left atrial pressure which in turn partially breaks the amount of regurgitation The increased atrial volume when transmitted across the mitral valve produces a better left ventricular filling It has been shown in models that the increase in the viscosity of the blood the celerity of the ventricular contraction and the lateral pressure effects on the mitral valve reduce the amount of regurgitation[5] On the other hand an increase in the aortic valve resistance systemic resistance or the size of the regurgitant orifice will favour the amount of mitral regurgitation Influence of cardiac rate on the amount of regurgitation is that the slower the rate the greater the regurgitation

RIGHT SIDED PRESSURES

The discussion of the right sided pressures here is based on the following two points

1 With significant regurgitation only mild to moderate mitral

stenosis can exist because severe stenosis with severe regurgitation is mechanically incompatible Generally the mitral valve area when associated with significant regurgitation does not go below 1 5 to 2 cm

ii A moderate amount of regurgitation of blood with a mitral valve area of 2 cm enlarges the left atrium but does not significantly raise the PC pressure any more than similar degree of mitral stenosis would do and hence it does not throw undue stress on the right ventricle However a severe degree of regurgitation when associated with a moderate stenosis can create significant pulmonary hypertension and stress the right ventricle enough to produce pulmonary oedema and/or peripheral circulatory failure of a very severe and ultimately fatal degree No such degree of regurgitation was studied in the present series

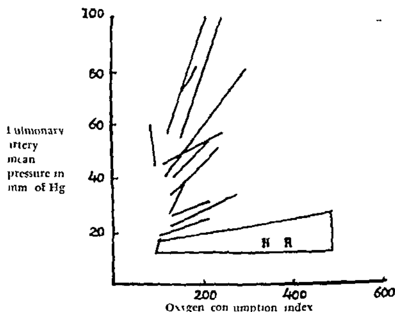
Right atrial mean pressure In the absence of congestive failure the right atrial mean pressure is found to be normal or near normal

Right ventricular diastolic pressure This is found to be O unless the right ventricle has started failing

Right ventricular systolic and mean pressure In a mild degree of mitral stenosis when associated with a moderately severe degree of mild regurgitation as was observed in some of our cases the right ventricular pressures were almost normal or somewhat elevated provided the pulmonary arteriolar resistances are normal On the other hand the right ventricular pressure was markedly elevated when a moderate degree of mitral stenosis was associated with an insignificant degree of mitral regurgitation Thus the right ventricular pressure is mainly regulated by the degree of mitral stenosis and the pulmonary arteriolar resistances The mitral regurgitation probably plays a relatively small role in raising this pressure The magnitude of the right ventricular pressure will be affected to a large measure by the degree of obstruction it has to overcome at the pulmonary arterioles and the mitral valve The pressure is affected perhaps to a lesser degree by the left ventricular failure or the extent of regurgitation

PULMONARY HYPERTENSION

Pulmonary arterial pressure The pulmonary arterial pressure was observed to rise according to the degree of the mitral stenosis During exercise the pulmonary arterial mean pressure behaviour in relation to the oxygen index arterio venous oxygen difference and the cardiac index has been shown below in the graphs



Oxygen consumption index

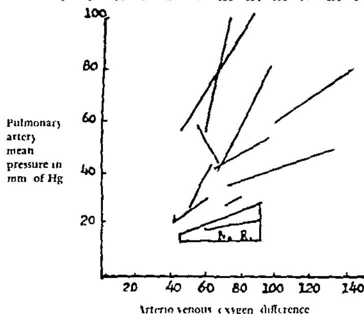
Figure 46

Predominant mitral insufficiency

Graph showing the relation ship of the pulmonary artery mean pressure with the oxygen consumption index

N R Normal Range adapted from L Dexter et al Jour App Phy 1951

Case Nos 151 152 153 251 451 452 453 454 455 456 459 462 (Appendix)



Arterio venous oxygen difference

Figure 47

Predominant mitral insufficiency

Graph showing the relationship of the pulmonary artery mean pressure with the arterio venous oxygen difference

N R Normal Range (adapted from L Dexter et al Jour Appl Phy 1951)

Case Nos 151 152 153 251 451 452 453 454 455 456 459 462 (Appendix)

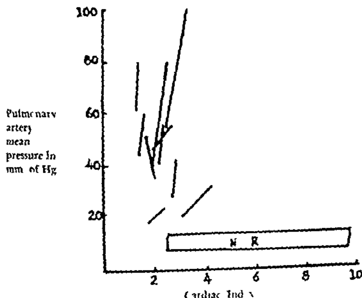


Figure 48

(Predominant mitral insufficiency)

Graph showing the relationship of the pulmonary artery mean pressure with the cardiac index

N R Normal Range (adapted from J P Hickman & W H Cargill J C Invest 1947)

Case Nos 151 152 153 2 1 451 4 2 4 3 4 4 3 1 ((Appendix)

(Compare with figures 19 20 and 21)

There was no significant difference observed in the general behaviour of the pulmonary arterial mean pressure in pure mitral stenosis (Refer to figures 19 20 and 21) and that in predominant mitral regurgitation (Refer to figures 46 47 and 48) but during exercise it is expected that the elevation in the pulmonary arterial mean pressure will be higher in cases of pure mitral stenosis than in cases of predominant mitral insufficiency

PULMONARY ARTERIAL WEDGE PRESSURE

The pulmonary arterial wedge mean pressure depends upon the following —

i Mitral Valve Area

Smaller the mitral valve area higher will be the pulmonary arterial wadge pressure

ii The mitral valve flow during the diastolic filling period

The mitral valve flow here represents the total left ventricular inflow or the total left ventricular output including the

forward flow into the aorta and the retrograde flow through the mitral valve. The total left ventricular inflow depends on the duration of the diastolic filling period and the degree of mitral disease while the total left ventricular outflow depends upon the efficiency of the ventricle.

Total mitral valve flow =

systemic output + regurgitated blood during systole

The amount of regurgitated blood forms generally a fraction of the total left ventricular output and therefore will affect the PW pressure fractionally. In predominant mitral regurgitation the systemic output was observed to be relatively smaller than in the similar degree of mitral stenosis. For a certain mitral valve area the effect of the regurgitation is to cut down the aortic outflow from what would have otherwise occured in pure mitral stenosis with the same mitral valve area.

The relationship of the PW pressure with the cardiac index was not different from that in pure mitral stenosis.

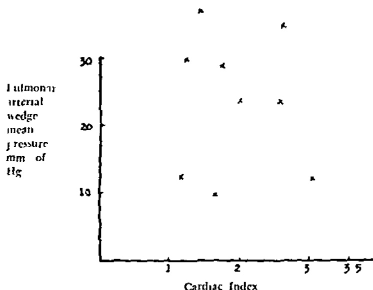


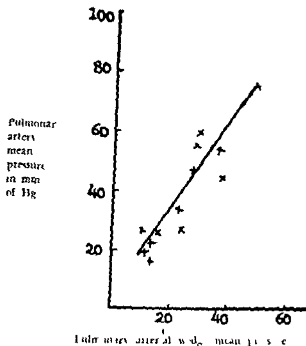
Figure 19

Predominant mitral insufficiency

Graph showing the lack of correlation between the cardiac index and the pulmonary arterial wedge mean pressure

Case Nos [illegible] (Appendix)

Also the behaviour of the PW pressure in relation with the pulmonary artery pressure was not significantly different.



Pulmonary arterial wedge mean pressure

Figure 30

Predominant mitral insufficiency

Graph showing the definite relationship of the pulmonary artery mean pressure with the pulmonary artery wedge mean pressure

Case No [illegible] 4,8 [illegible] (Appendix)

Considering the height and the very short duration of the regurgitant *v* wave in relation with the total duration of the cardiac cycle it is believed that the degree of regurgitation as much as seen in the present series does not affect the pressures in the pulmonary circuit to any great extent The PW pressure appeared to be mainly due to the degree of the mitral stenosis In accordance with that the PW pressure is not found unusually higher than what can occur from the similar degree of pure mitral stenosis alone The amount of regurgitation in the range observed in this series may at its maximum contribute to the elevation of the PW mean pressure by about 5 to 6 mm Hg

PAm—PWm GRADIENT

As has been pointed out before with a relatively small degree of regurgitation as much as is seen in the present study the pressure in the pulmonary circuit is raised to a small extent from the values that can be obtained in a comparable degree of pure mitral stenosis The extent of the small rise due to the regurgitation in both the PAm and the PWm below the pulmonary oedema level will depend upon the pulmonary arteriolar resistance at rest

Thus the gradient PAm — PWm depends upon mainly the pulmonary arteriolar resistance and not on the amount of regurgitation

The relationship between the PAm and the PAm — PWm gradient shows that all the cases except one having PAm higher than 40 mm Hg had the gradient PAm — PWm greater than 10 mm Hg

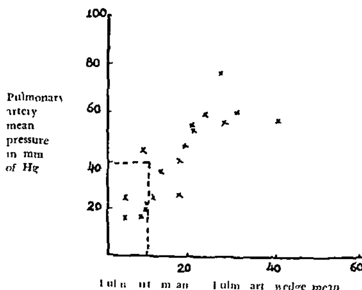


Figure 51

(Predominant mitral insufficiency)

Graph showing the relationship of the difference between the pulmonary artery mean pressure and the pulmonary artery wedge mean pressure with the pulmonary artery mean pressure

Case No 1 1 1 1E 1 2 1 3 1,3F 4 1 4 2 4 2F 4,3 4 4 4,3 4 6 4 6E 45, 4 8 4 8E 4 9 46 462 (Appendix)

The general behaviour of the gradient of PAm — PWm in relation with the PWm is just the same as in pure mitral stenosis

Thus the other factors affecting the PAm — PWm gradient are no different from pure mitral stenosis

OXYGEN CONSUMPTION
ARTERIO—VENOUS OXYGEN DIFFERENCE
CARDIAC OUTPUT

Oxygen consumption —

Tissue demand for oxygen remains the same irrespective of regurgitation Oxygen consumption at rest in cases of predominant regurgitation has been found within the normal range

During exercise oxygen demand goes higher depending upon the degree of exercise However it is the impression that a greater degree

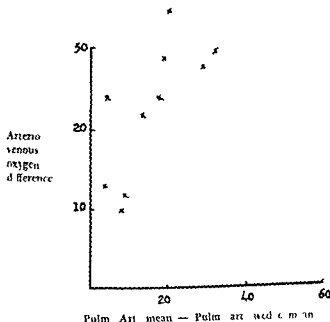


Figure 5

Predominant mitral insufficiency

Graph showing the relationship of the difference between the pulmonary artery mean pressure and the pulmonary arterial wedge mean pressure with the pulmonary arterial wedge mean pressure

Case No 131 132 133 4 1 43 433 [illegible] (Appendix)

of voluntary restriction in effort is motivated on the part of the patients with predominant regurgitation

Arterio venous oxygen difference —

An arterio venous oxygen difference at rest is usually found slightly higher than pure mitral stenosis in order to make a reciprocal adjustment with the diminution in systemic output During exercise the cardiac output showed a tendency to rise relatively less than in pure mitral stenosis as compared to the oxygen consumption Tissue extraction of oxygen from the available blood goes inordinately high resulting in a high arterio venous difference

Cardiac output and Cardiac index —

The total left ventricular output is greater than the effective cardiac output as a part of it is directed back through the mitral valve In a compensated cardiac patient the cardiac output (or effective cardiac output or the aortic output or the systemic output) has to be equal to the right ventricular output The average cardiac or cardiac index in this group at rest is slightly lower than seen in a similar degree of pure mitral stenosis

Arterio venous oxygen difference

100
80
60
40
20

N R E

N R

200 400 600

Oxygen Consumption Index

Figure 53

Predominant mitral insufficiency

Graph showing the relationship of the Arterio-venous oxygen difference with the oxygen consumption index at rest and during exercise

Disproportionate elevation of A – V difference during exercise is seen

N R Normal range

N R E Normal range during exercise

Case No [illegible] 6 [illegible] 9 [illegible] 450 [illegible] (Appendix)

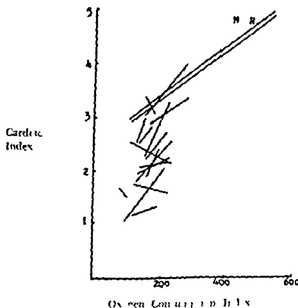
The maintenance of the demanded cardiac output is attempted probably by the complex mechanism in the nervous system. However the systemic output can be affected in double mitral lesion by the following factors —

i Mitral valve stenotic factors
ii Mitral valve regurgitant factors
iii Left ventricular efficiency
iv *Right ventricular efficiency*
v Systemic resistance
vi Total pulmonary resistance

Mitral valve stenotic factors These factors reduce the systemic output diminishing *the total left ventricular inflow The total left ventricular inflow* is affected by the mitral valve stenotic factors can be discussed by considering the formula —

$$\text{Mitral valve stenotic area} = \frac{\text{Mit Val Flow (Total L V Inflow)}}{\text{Dias Fill Period} \times 31 \times \sqrt{\text{PWm} - 5}}$$

$$\text{or Mitral Valve Flow} = \text{Mitral Val Steno Area} \times \text{Dias Fill Period} \times 31 \times \sqrt{\text{PWm} - 5}$$



Oxygen Consumption Index

Figure 4

Predominant mitral insufficiency

Graph showing the relationship of the cardiac index with the oxygen consumption index at rest and during exercise. Disproportionately smaller rise in cardiac index during exercise.

N R Normal Range

Case No 151 152 154 240 2[illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] 41[illegible] 46 (Appendix)

Now the systemic output (the cardiac output) depends upon the total left ventricular inflow which in turn depends upon the following three factors

a Mitral valve stenotic area Although the mitral valve stenotic area does not directly affect the systemic output but it restricts the total left ventricular stroke volume and is unable to compensate for the regurgitation To that extent therefore the degree of mitral stenosis contributes to the reduction in the systemic output

b Diastolic filling period No definite proof is available as to be able to decide whether the diastolic filling period is any different in patients with the double mitral lesions from those having pure mitral stenosis of similar degree

c PW pressure As discussed previously the PW pressure in double mitral defect does not rise to unusual degree of pure mitral stenosis The height of PW pressure is mainly guided by the severity of stenosis The accomplished in

crease in the magnitude of the PW pressure due to the associated regurgitation is not adequate enough to elevate the total left ventricular inflow in order to compensate for the amount regurgitated. During exercise in patients with double mitral lesions the degree of mitral stenosis affects the total left ventricular inflow in the same way as in patient with similar degree of pure stenosis.

During tachycardia of exercise the diastolic filling period decreases and the PW pressure rises depending upon the degree of stenosis. However this rise in PW pressure is not enough to fully compensate the fall in the diastolic filling period. The total left ventricular inflow therefore during the ventricular diastole will be reduced. There is reduction in systemic output in proportion to the overall reduction in the total left ventricular inflow. Thus much reduction in the systemic output is the contribution of the mitral stenosis during exercise in patients with the double mitral lesion.

ii. **Mitral valve regurgitant factors.** From the total volume of left ventricular blood certain amount of blood is deviated through the mitral valve by way of regurgitation. The systemic output therefore suffers a reduction by the amount regurgitated.

Systemic output = Total L. V. inflow — The amount regurgitated.

The total left ventricular inflow (same as the total left ventricular *output*) is mainly controlled by the total mitral valve area during the ventricular diastole likewise the amount regurgitated by the mitral valve *regurgitant* area during the ventricular systole. The factors affecting the amount of blood regurgitating can be best discussed by considering the following *formula*.

$$\text{M. V. Regurgitant Area} = \frac{\text{The amount of blood regurgitated}}{\text{S E P} \times 36 \times \sqrt{\text{Bra. Art. Sys. mean} - \text{PAm}}}$$

$$\text{OR Amt. Regurgitated} = \text{M. V. Reg. Area} \times \text{S E P} \times 36 \times \sqrt{\frac{\text{Bra. Art.}}{\text{Sys. mean} - \text{PAm}}}$$

Thus the amount of blood regurgitating depends upon the following.

1. The mitral valve regurgitant area. The amount of regurgitating blood mainly depends upon the mitral valve regurgitant area. It has been shown[4] that the volume of blood regurgitating has almost linear correlation with the mitral valve regurgitant area.

b Systolic ejection period This period has not been observed to be any different in double mitral lesions from the similar degree of mitral stenosis

c Transvalvular pressure gradient The transmitral pressure gradient (between the left atrium and left ventricle) is high because of elevated left atrial pressure unless some other factor is associated which raises the peripheral resistance and thereby the left ventricular systolic pressure

During exercise the transvalvular pressure gradient quite large at rest a small change in it does not alter the regurgitating volume of blood significantly The tachycardia of effort increases the systolic ejection period considerably and this in turn raises the regurgitant flow as well as the systemic output But the rise in the systemic output occurs to a lesser degree than what can occur with a similar degree of pure mitral stenosis because of the undue loss from the total left ventricular volume by way of regurgitation Although no apparent relationship could be found between the systemic output and the amount of regurgitation the patients with double mitral defects are affected much more adversely in their systemic output during effort

iii **Left ventricular efficiency** This is equally important in the maintenance of the systemic output The left ventricular capacity is higher in the *presence* of mitral insufficiency Within the physiologic limits increased left ventricular pumping ability is not adequate enough to compensate completely Varying degree of reduction in the systemic output therefore is observed in the present series The degree of regurgitation increases the work load of the left ventricle markedly and therefore in the face of increased demand from the left ventricle the left ventricular work against pressure increases

The left ventricle anatomically and physiologically is the most efficient chamber and can stand up to great demand Considerable degree of increased load can be taken up by the left ventricle without raising the left ventricular diastolic pressure The time factor during which mitral regurgitation occurs over a long period gives enough time to the left ventricle to adjust to the new demand The mitral regurgitation of sudden onset is entirely a different problem Rarely the patients during mitral valvotomy die because of the inability of the left ventricle to cope with the sudden creation of mitral regurgitation resulting into the systemic output diminished below levels compatible with life Nevertheless when the left ventricle starts failing because of the active rheumatic carditis or its inability to take the load the left ventricular dis

tolic pressure goes high and to that extent the PW pressure is elevated which may at a point result into pulmonary oedema

iv Right ventricular efficiency A pure mitral stenosis tends to limit the left ventricular inflow sparing the left ventricle at the expense of the right ventricle but in double mitral lesions the demand from the right ventricle for the ejection of blood is less than in pure mitral stenosis The right ventricular output is equal to the systemic output However the right ventricular pressure work is lower in cases of double mitral defect than in cases of similar degree of pure mitral stenosis although the pulmonary pressures may be slightly higher due to the regurgitation

v Systemic and aortic valvular resistances When the total systemic resistance is high for one reason or the other a part of the left ventricular energy is spent in overcoming the high friction due to the resistence The systemic output therefore is more limited Besides with higher systemic resistence the systolic ejection period is prolonged and the trans valvular systolic pressure gradient is raised These will lead to the increase in the amount of regurgitating blood When the systemic resistance is diminished the same amount of left ventricular energy becomes more productive in terms of systemic output If the mitral insufficiency is associated with the aortic stenosis the left ventricle has to overcome this obstruction and therefore the regurgitant orifice gets the preference over the aortic orifice in the distribution of blood from the left ventricle The aortic output is diminished to that extent

vi **Total pulmonary resistance** The total pulmonary resistance acts as a check to the right ventricular output In double mitral lesion the elevation of the total pulmonary resistence can be attributed to the following factors

a Narrowed pulmonary vascular bed

b Obstruction due to the mitral valve narrowing

c Increased blood volume in the left atrium due to the back flow through the regurgigating mitral valve

All these three factors are active in combined mitral defect while the first two only in pure mitral stenosis The right ventricular output and hence the systemic output are curtailed relatively more in the presence of regurgitation

Conclusion

Mitral stenosis by itself checks the systemic output by limiting the total left ventricular inflow With the given degree of mitral stenosis

the systemic output is further reduced by the mitral regurgitation provided other factors discussed above remain the same As the stenosis increases the total left ventricular inflow decreases As the regurgitation increases the systemic output decreases However marked stenosis and marked insufficiency are anatomically impossible in the same patient

Thus as far as cardiac output (systemic output) is concerned both mitral stenosis and mitral regurgitation when associated have a tendency to worsen the effect of the other

VASCULAR RESISTANCES
RIGHT VENTRICULAR PRESSURE WORK
LEFT VENTRICULAR PRESSURE WORK

Pulmonary arteriolar resistance In general the pulmonary arteriolar resistance is not observed to be higher than those with similar degree of pure mitral stenosis

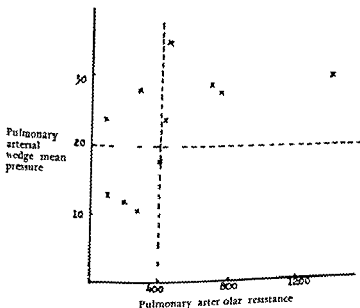


Figure 55
Predominant mitral insufficiency
Graph showing the relationship of the pulmonary arteriolar resistance with the pulmonary arterial wedge mean pressure
Case Nos 152 226 229 230 431 438 439 4 1 45 4 6 4 7 459

The relationship of the pulmonary arteriolar resistance with PWm is shown in figure 55 The patients having pulmonary arteriolar resistance below 400 and PWm below 20 had mitral regurgitation without left ventricular enlargement fluoroscopically The patients having their

PWm above 20 had mitral regurgitation with left ventricular enlargement fluoroscopically Of the latter group those having their pulmonary arteriolar resistances above 400 also had fair degree of mitral stenosis of some duration It is believed that enlarged left ventricule is likely to have the diastolic pressure higher than O and therefore contributes to elevating the PWm pressure The associated mitral stenosis as well as the state of left ventricular myocardium appeared to be mainly responsible for the increased pulmonary arteriolar resistances

If the regurgitation is severe the left ventricular failure results into significantly elevated left ventricular diastolic pressure In that case the PWm pressure has got to be elevated in order to pump against the high left ventricular diastolic pressure which in turn will raise the question of protection of pulmonary capillaries and thereby augmenting the pulmonary arteriolar resistances The elevated pulmonary arteriolar resistances in double mitral lesion can be attributed to the following

a Associated mitral stenosis
b Back thrust in the left atrium due to regurgitation
c The state of the left ventricular myocardium (or the degree of left ventricular failure)

The pulmonary arteriolar resistance was observed to have no relation with the amount of regurgitating blood or the systemic output

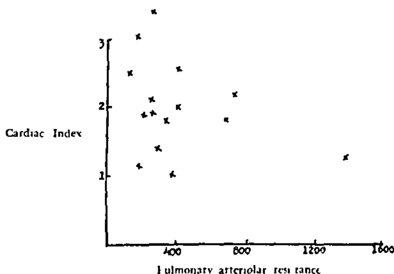


Figure 36

Predominant mitral insufficiency

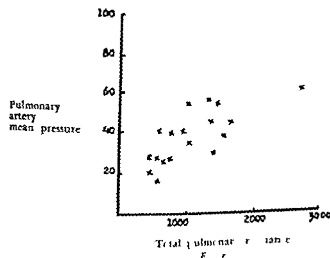
Graph showing the relationship of the pulmonary arteriolar resistence with the cardiac index

Case Nos 151 152 2 6 229 30 451 4 2 4 3 451 4 5 4 6 4 4 8 4 4 460 46

Unlike pure mitral stenosis lack of any correlation is observed between the pulmonary arteriolar resistance and the cardiac index (systemic index)

Total pulmonary resistance Although the mitral regurgitation does not significantly alter the pressures in the pulmonary circuit it diminishes effectively the systemic output The pulmonary pressure is not able to accomplish the systemic output as much as it can in case of pure mitral stenosis The total pulmonary resistance appeared to be higher than in similar degrees of pure mitral stenosis

The systemic output showed a tendency to drop as the total pulmonary resistance increased The pulmonary artery mean pressure showed a tendency to rise as the total pulmonary resistance increased



Total pulmonary resistance

Predominant mitral insufficiency

Graph showing the relationship of the total pulmonary resistance with the pulmonary arterial mean pressure

Case No [illegible]

The correlation is not as good as in pure mitral stenosis The right ventricle here is working against the higher resistance Much of the right ventricular pressure work is spent in maintaining the pressure on the mitral valve rather than in propelling the blood

Mitral resistance The mitral resistance was observed to be slightly higher than in a similar degree of pure mitral stenosis With a double mitral lesion the mitral valve offers some resistance to the flow both during the left ventricular diastole and the left ventricular systole During the ventricular systole the regurgitating blood in the left atrium main

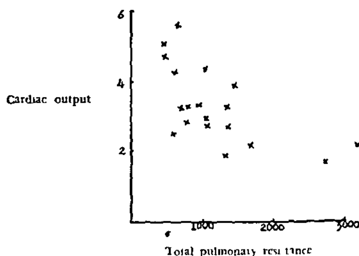


Figure 58

Predominant mitral insufficiency

Graph showing the relationship of the total pulmonary resistence with the cardiac output

Case Nos 151 152 154 155 226 227 29 31 451 452 4 3 454 455 456 4 7 4 9 460 461 4(

tains the resistance offered to the flow from the pulmonary circuit towards the left atrium

The PWm showed a general tendency to rise up to the pulmonary oedema level as the mitral resistance increased The systemic output

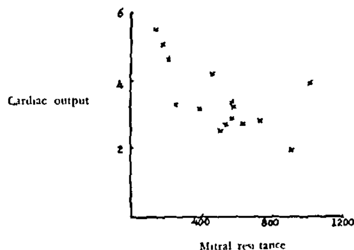


Figure 59

Predominant mitral insufficiency

Graph showing the mitral resistence in relation to the cardiac output

Case Nos 151 152 154 155 226 2 9 231 451 4 2 453 454 455 4 6 457 459 460 462

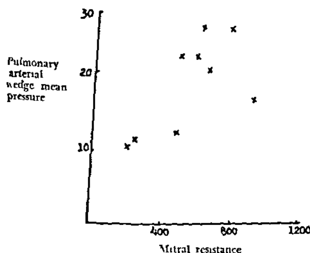


Figure 60

Predominant mitral insufficiency

Graph showing the relationship of the mitral resistence with the pulmonary arterial wedge mean pressure

Case No 151 152 226 29 230 451 4 453 4 4 4 456 45 45

showed a tendency to drop as the mitral resistance increased up to a certain limit

Mitral valve resistance Mitral valve resistance here is much less Major part of the mitral resistance is left ventricular resistance because of high diastolic pressure in the failing left ventricle

Systemic resistance The systemic resistance appeared to be further elevated from those observed in a similar degree of mitral stenosis The rise in the systemic resistance is roughly parallel to the fall in the systemic index

The general behaviour of the systemic index is similar to that in pure mitral stenosis

RIGHT VENTRICULAR PRESSURE WORK

Right ventricular pressure work = Cardiac Index x (PAm — RAm) x constant

Although the pulmonary pressure is elevated in predominant mitral insufficiency the systemic index is reduced as compared to that in pure mitral stenosis

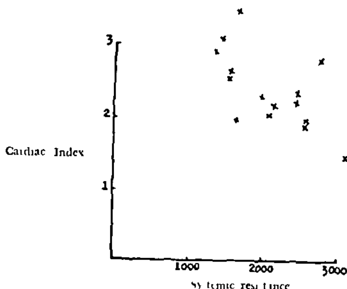


Figure 11

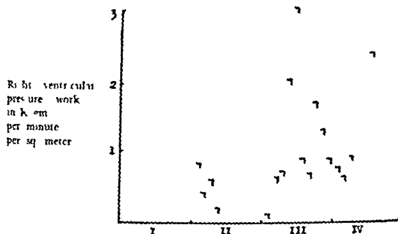
Predominant mitral insufficiency

Graph showing the relationship of the systemic resistence with the cardiac index
Case Nos 151 15 154 155 226 9 90 4 1 45 4 3 454 455 4 6 458 459
160 461 16

The cardiac index or the systemic index which is equal to the right ventricular index) is further lowered from that in equal degree of mitral stenosis The right ventricular pressure work performed therefore was less in this group The left ventricle here shared the brunt of the burden Among the patients in group III[2] according to the functional classification of the New York Heart Association 9 out of 13 showed their right ventricular pressure work to be less than one as compared to 3 out of 9 in pure mitral stenosis

The right ventricle is partially spared partly because of the diminished pulmonary flow and partly because of the relatively low pulmonary arteriolar resistance A significant portion of the right ventricular pressure work is expended in keeping a check on the mitral regurgitation during the left ventricular systole and the rest of the right ventricular pressure work is used up in maintaining an adequate pressure head on the mitral valve in order to fill up the left ventricle during the diastole

Another way of looking at it is that the high left atrial pressure is partly maintained by the regurgitation Depending upon the amount of regurgitation the demand from the right ventricle is increased Whenever the amount of regurgitation increases the right ventricular pressure work also tends to increase



Symptomatic grades according to the criteria of New York Heart Association

Figure C

(Predominant mitral insufficiency)

Graph showing the right ventricular pressure work in different symptomatic grade
(Four grades according to the criteria of New York Heart Association)

Case Nos 13 152 154 15 22 22 2 23 41 42 43 4 47 48 49
160 41 40

LEFT VENTRICULAR PRESSURE WORK

The part of the left ventricular pressure work is spent in ejecting the blood through the aortic valve and the part of it in regurgitating through the mitral valve The total left ventricular output is greater than the cardiac output in normals unless the left ventricle is failing If the systemic resistance is normal and if the aortic valve is normal the left ventricular pressure work depends upon the total left ventricular output or the total left ventricular inflow which varies as the mitral valve area Thus the left ventricular pressure work is greater with the greater total valve area With the severe degree of regurgitation more than 50% of the left ventricular pressure work can be used up in regurgitating the blood through the mitral valve

The effective left ventricular pressure work in respect to propelling of blood into the aorta depends upon the degree of regurgitation and the magnitude of the systemic resistance

SYSTOLIC EJECTION PERIOD
MITRAL VALVE REGURGITANT AREA

Systolic ejection period The regurgitation occurs during the left ventricular systole The systolic ejection period is measured generally from the brachial artery tracing The regurgitation really starts slightly before the opening of the aortic valve and continues slightly after the closure of the aortic valve These time differences are only 0 01 to 0 02

seconds particularly in cases of high left atrial pressures Measurement of the systolic ejection period from the brachial artery tracing therefore will not be affected significantly

During exercise as the diastolic filling period decreases the systolic ejection period increases The changes in the left atrial pressure and other factors changing during effort will counterbalance the increase in systolic ejection period

The role of the systolic ejection period will be evident by looking at the formula for the mitral valve regurgitant area —

$$\text{Mit Val Reg Area} = \frac{(\text{M V A} \times \text{D F P} \times 97\sqrt{\text{PWm} - 5}) - \text{Cardiac output}}{\text{S E P} \times 36 \times \sqrt{\text{LV Sys mean} - \text{PWm}}}$$

(Please refer to part I chapter 7)

Mitral valve regurgitant area The consideration of the effect of varying magnitude of mitral valve regurgitant area on the other aspects of the dynamics have been done in the course of the discussion previously

Conclusions

1 The size of the mitral valve regurgitant area by itself does not elevate the pressures anywhere in the pulmonary circuit to any degree of consequence

2 The effective cardiac output or the systemic output is reduced greatly by the degree of regurgitation

3 The right ventricular pressure work is less than that in a similar degree of the mitral stenosis

4 The left ventricular pressure work is more in regurgitation as compared to that in a similar degree of mitral stenosis

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CHAPTER IV

HAEMODYNAMIC BASIS OF SYMPTOMS AND SIGNS OF MITRAL DISEASE

Many of the symptoms and the clinical signs of the mitral disease are the result of the pulmonary congestion or systemic congestion or both. Amongst the patients with similar disease one may have more of one symptom than the other. The reason for the precedence of one symptom over the other with similarly deranged haemodynamics is not always clear. Nevertheless there is always a physiologic basis about the causation of any one symptom.

An attempt has been made below to correlate the clinical picture with the altered haemodynamics —

Symptoms of Pulmonary Congestion (Exertional dyspnoea, orthopnoea and paroxysmal nocturnal dyspnoea)

This group of symptoms is more marked in predominant mitral stenosis than in mitral insufficiency. *It is difficult to evaluate this* subjective group of symptoms. Although no close correlation can be expected between these essentially subjective feeling on the part of the patients and the underlying physiologic derangement, an attempt has been made to show the general tendency of the relationship of these symptoms with the altered physiology.

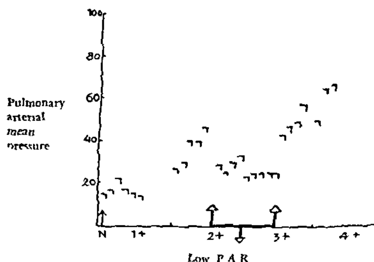
Role of pulmonary artery pressure. The pulmonary arterial mean pressure is observed to have a rough correlation with the symptoms. The symptoms of the pulmonary congestion have been graded as follows —

Grade I — Slight exertional dyspnoea without orthopnoea or paroxysmal nocturnal dyspnoea.

Grade II — Marked exertional dyspnoea with slight orthopnoea and paroxysmal nocturnal dyspnoea.

Grade III — Dyspnoea on the slightest exertion during the routine activities with two pillows orthopnoea and occasional paroxysmal nocturnal dyspnoea.

Grade IV — Apparent dyspnoea even at rest, three to four pillows orthopnoea and frequent attacks of paroxysmal nocturnal dyspnoea.



Low P A R

Figure 63

Grades according to symptoms of pulmonary congestion

Predominant mitral stenosis

Graph showing the relationship of the pulmonary arterial mean pressure with the grades according to the symptoms of pulmonary congestion

A group of cases with low P A R (Pulmonary arteriolar resistances) has been shown with arrows

N Normal *Four grade 1 2 3 4*

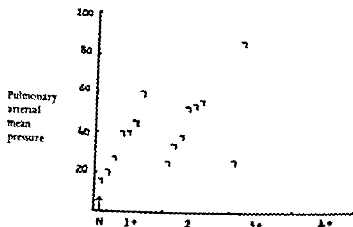
Cases Nos 101 103 104 105 106 1[illegible] 108 109 110 111 112 113 114 115
116 118 [illegible]01 [illegible]03 [illegible]04 [illegible] 403 40[illegible] 408 409 410 41[illegible] 41[illegible] 418

Figure 63 shows that the group of patients having marked symptoms of pulmonary congestion with moderately elevated PAm had low pulmonary arteriolar resistance

It is noted from the figure 63 and 64 that in mitral stenosis the symptoms of the pulmonary congestion increase from 1 to 4 plus as the pulmonary arterial mean pressure rises whereas in predominant mitral insufficiency relatively higher pulmonary arterial mean pressures are seen for the milder symptoms of 1 to 2 plus pulmonary congestion

	Range of PAm in mitral stenosis in mm of Hg	Range of PAm in predominant mitral insufficiency in mm of Hg
Grade I	13 to 22	22 to 61
Grade II	26 to 46	26 to 56
Grade III & IV	23 to 64	—

Table showing the comparative figures in cases of predominant mitral stenosis and predominant mitral insufficiency as related to the symptoms of pulmonary congestion



Grades according to symptoms of pulmonary congestion

Figure 64

Predominant mitral insufficiency

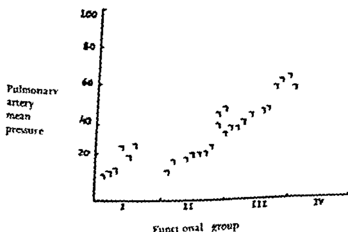
Graph showing the relationship of the pulmonary artery mean pressure with the grades according to the symptoms of pulmonary congestion

N Normal Four grades 1 2 3 4

Case Nos 13 124 122 431 42 433 434 [illegible] 458 459 460 461 4[illegible]

The pulmonary arterial mean pressure and the functional groups —

(The functional groups are according to the criteria of New York Heart Association)



Functional group

Figure 65

Predominant mitral stenosis

Graph showing the relationship of the pulmonary arterial mean pressure with the functional classification in four groups according to the criteria committee of the New York Heart Association

Case Nos 101 10[illegible] 103 104 105 106 107 108 1[illegible] 110 1[illegible] 114 11[illegible] 116, 118
101 10[illegible] 103 104 105 105 10[illegible] 108 1[illegible] 110 1[illegible] 11[illegible] 118
201 203 204 205 105 105 108 [illegible]

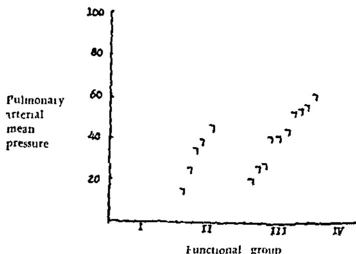


Figure 66

Predominant mitral insufficiency

Graph showing the relationship of the pulmonary artery mean pressure with the functional classification in four groups according to the criteria committee of the New York Heart Association

Case No [illegible] 453 454 455 451 45 45[illegible] 45[illegible] 45[illegible] 456 457 458 459 460 461 462

Good correlation is observed in mitral stenosis group only because in mitral insufficiency the function of the patient is limited by further reduction of the cardiac index

	Range of PAm in mitral stenosis in mm of Hg	Range of PAm in predominant mitral insufficiency in mm of Hg
Group I	13 to 28	——
Group II	17 to 50	17 to 46
Group III & IV	10 to 68	22 to 85

Role of pulmonary arterial wedge mean pressure —

The general tendency of increasing dyspnoea with the rising PWm pressure is noted in the figure 67 In the patients below the oblique line with 3 to 4 plus symptoms as shown in the figure had relatively low pulmonary arteriolar resistances This group therefore is believed to be having disproportionately more symptoms with comparatively low PWm pressure

Although a small group the patients with predominant mitral insufficiency showed that with high PWm symptoms of pulmonary congestion were relatively mild It is interesting to point out that this particular group having high PWm had only moderately elevated PAm and the gradient PAm — PWm was relatively smaller

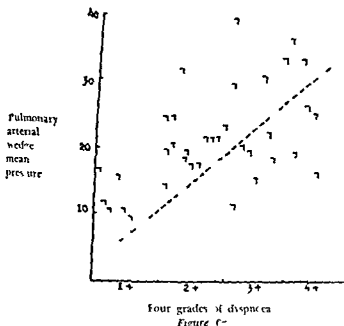


Figure 6–

Predominant mitral stenosis

Graph showing the relationship of the pulmonary arterial wedge mean pressure with the four grades of dyspnoea

Cases falling below the oblique line [illegible] + dyspnoea had elevated pulmonary arteriolar resistances

1+ Slight Symptoms on unusual effort

2+ Moderate Symptoms on ordinary exertion

3+ Considerable Symptoms on necessary routine activities

4+ Incapacitating Total disability

Cases Nos 103 104 106 107 108 109 [illegible] 111 [illegible] 113 114 118 [illegible] 119
131 [illegible] 201 [illegible] 404 408 409 410 414 415
[illegible] 418 419 [illegible]

A wedged catheter in a pulmonary arteriole reflects the cyclic changes in the left atrium [1, 2, 3, 4, 6, 8, 9, 13, 5, 16, 22]. Similarly a wedged catheter in a pulmonary vein reflects the cyclic changes in the pulmonary artery [2]. The actual pressure in the pulmonary capillaries therefore should be somewhere in between PAm and the left atrial mean pressure. In mitral insufficiency the PWm and therefore the left atrial mean pressure is partly the result of the left ventricular systolic mitral regurgitant stream. The pulmonary capillaries therefore get a part of its pressure head from the left ventricle via the left atrium and the rest of the pressure head is derived from the right ventricle via the pulmonary arteries. It is hypothesized therefore that the actual pressure in the pulmonary capillaries in mitral insufficiency is closer to the left atrial mean pressure. Where as in mitral stenosis the pulmonary capillaries derive its pressure head from the right ventricle via the pulmonary arteries in its entirety and therefore the pulmonary capillary pressure should be

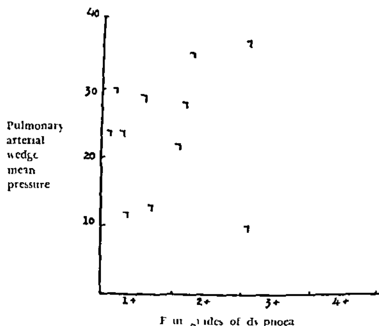


Figure 68

Dominant mitral insufficiency

Graph showing the relationship of the pulmonary arterial wedge mean pressure with the four grades of dyspnoea

Case No 15 13 41 42 43 44 45 46 4[illegible] 49

closer to the pulmonary artery mean pressure Therefore for a fixed PWm recorded the actual pressure in pulmonary capillaries in mitral stenosis will be higher than that in mitral insufficiency Thus for an equivalent amplitude of PWm pressure or the left atrial mean pressure the symptoms of the pulmonary congestion are more in mitral stenosis than in mitral insufficiency

Role of mitral valve area —

It is now well known that the symptomatology in mitral stenosis is mainly the result of two factors namely the narrowing of the mitral opening and that of the pulmonary arterioles

Shown in figure 69 is the relationship of the symptoms of the pulmonary congestion with the mitral valve area The result is more or less similar to that obtained by Gorlin [14] The patients with a relatively big mitral valve area of 1·05 to 2 cm and the pulmonary arteriolar resistance below 160 had only mild or 1 plus symptoms of the pulmonary congestion As the mitral valve area diminished from 1·05 to 0·5 cm² causing fairly severe stenosis the pulmonary arteriolar resistance rises slightly from 110 to 440 This is considered as the transition stage when the mitral valve is pretty tight but the pulmonary arterioles did not get a chance enough to increase

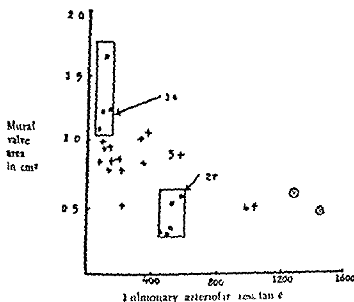


Figure 69

Predominant mitral stenosis

Graph showing the relationship of the pulmonary arteriolar resistance and the mitral valve area in cm² with the symptoms of pulmonary congestion (symptoms of P C) 1 2 3 and 4 +

Case Nos 10 103 104 106 107 108 109 110 111 112 115 118 201 203 20 403 405 408 409 410 415 417

the resistance for its own protection The symptoms of pulmonary congestion therefore increase quickly to 3 plus and the patients in this stage may get paroxysmal dyspnoea and the pulmonary oedema too Following this stage the course of the disease becomes steady and the mitral valve area remains around 0 5 cm² However the pulmonary vasculature gets time to build up the resistance and the pulmonary arteriolar resistance increases from 140 to 600 This is protecting the pulmonary capillaries and therefore some improvement in the symptoms (2 plus) of the pulmonary congestion occurs The elevated pulmonary arteriolar resistance increases the right ventricular load and the right ventricle begins to fail It is now a days clinically well recognised that in the course of the mitral stenosis leading to the pulmonary hypertension of post capillary origin there is temporary alleviation of the pulmonary symptoms with the onset of the right ventricular failure Such apparent improvement in the natural course of the disease sometimes is erroneously attributed to the therapeutic measures With the further progress of the disease, the pulmonary arteriolar resistance still further increases from 600 to 1440 and the signs of the right ventricular failure become more marked The cardiac output in this stage is further reduced and there is onset of easy fatigue and the dyspnoea together

The following table concludes the findings as related to the symptoms of the pulmonary congestion —

	Range of mitral valve area in cm²	Range of pulmonary arteriolar resistance
Grade I (1 plus)	1 05 to 2 0 cm²	Below 160
Grade II (2 plus)	0 5 to 1 05 cm²	440 to 600
Grade III & IV (3 to 4 plus)	0 5 or less	140 to 440 or 600 to 1 440

Paroxysmal nocturnal dyspnoea

All the patients having the history of paroxysmal nocturnal dyspnoea fell into the range of pulmonary arteriolar resistance of Grade II and IV i e between 140 and 440 or between 600 and 1440 and had mitral valve area of 1 plus minus 0 5 cm²

Orthopnoea

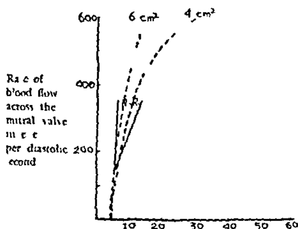
Orthopnoea generally occurs late in the course of the disease All the patients with history of orthopnoea fell into Grade IV In sitting posture the right atrial pressure and therefore the right ventricular output is lower This in turn lowers left atrial or the pulmonary capillary pressure resulting into diminished pulmonary congestion

PULMONARY OEDEMA

Haemodynamic basis of pulmonary oedema —The attacks of pulmonary oedema in mitral stenosis are much more frequent than in mitral insufficiency because left atrial mean pressure in mitral stenosis is higher than in mitral insufficiency of comparable degree and also left atrial pressure in mitral stenosis tends to rise more sharply during exertion The paroxysmal attacks of pulmonary oedema occurs in about 107 of all cases of mitral stenosis [27]

The understanding of the haemodynamic basis of the pulmonary oedema in mitral stenosis hinges around the concept of inter relations of several factors with one another namely the pulmonary arterial wedge pressure (pulmonary capillary pressure) rate of blood flow across the mitral valve and the extent of mitral stenosis If the required effective hydrostatic pressure in the pulmonary capillaries needed to maintain the necessary rate of flow across the mitral valve is higher than the assumed pulmonary oedema level transudation can occur and the actual pulmonary oedema may develop provided the high pressure is maintained for long enough time

The pressure flow relation at various valve sizes in pure mitral stenosis can best be understood from the curves below —



Pulmonary capillary pressure for a normal mitral valve area of 4 to 6 cm²

Figure

Theoretical

Graph showing the relationship of the rate of flow across the mitral valve in c c per diastolic second with the pulmonary capillary pressure for a normal mitral valve size of 4 to 6 cm²

(Adapted from Gorlin et al A H Jour 1951) (17)

N R Normal Range

(Although it is realized that the PW pressure is not exactly equal to the pulmonary capillary pressure it is believed that the PW pressure is a very close index of pulmonary capillary pressure)

The rate of flow across the mitral valve is the net amount of blood in c c flowing in the left ventricle during each diastolic second Normal range of mitral valvular flow considered is 150 to 250 c c When the mitral valve size is in the normal range of 4 to 6 cm² small rise in the PW pressure can force large amount of blood in the left ventricle

With a mitral valve size of about 2 4 cm² as shown in figure 71 normal mitral valve flow can be maintained at rest but the pulmonary reserve is lost The need for additional flow during effort may raise the pulmonary capillary pressure as reflected through the PW pressure to a level higher than the transudation level producing the pulmonary congestion

In a moderate stenosis with a mitral valve area of 1 4 cm² adequate mitral flow still can be maintained without raising the pressure to pulmonary oedema level No symptoms therefore may occur with these

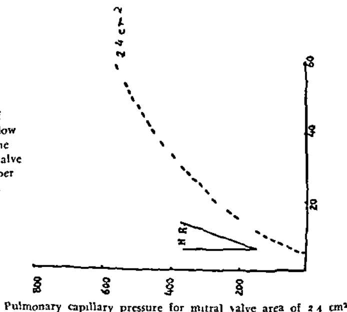


Figure 71

Theoretical

Graph showing the relationship of the rate of blood flow across the mitral valve in c c per diastolic second with the pulmonary capillary pressure for a moderate mitral stenosis of 2 4 cm² valve area

(Adapted from Gorlin et al A H Jour 1951) (17)

N R Normal Range

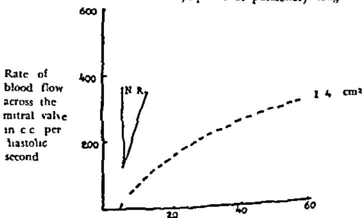


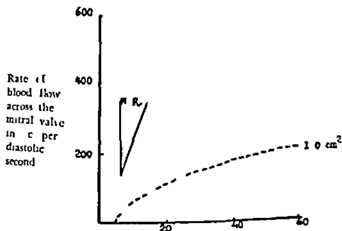
Figure 72

Theoretical

Graph showing the relationship of the rate of blood flow across the mitral valve in c c. per diastolic second with the pulmonary capillary pressure for a moderately severe mitral stenosis of 1 4 cm² valve area (17)
(Adapted from Gorlin et al A H Jour 1951)

N R Normal Range

people at rest Mild effort requiring the higher rate of flow may necessitate the pressure close to the pulmonary oedema level



Pulmonary capillary pressure for mitral valve area of 1 0 cm

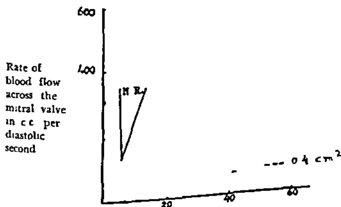
Figure 73

Theoretical

Graph showing the relationship of the rate of blood flow across the mitral in c c diastolic second with the pulmonary capillary pressure for a severe mitral stenosis of 1 cm² valve area

(Adapted from Gorlin et al A H Jour 1951) (17)

N R Normal Range



Pulmonary capillary pressure for mitral valve area of 0.4 cm²

Figure 74

Theoretical

Graph showing the relationship of the rate of blood flow across the mitral valve in c c per diastolic second with the pulmonary capillary pressure for a very severe mitral stenosis of 0 4 cm² valve area

(Adapted from Gorlin et al A H Jour 1951) (17)

N R Normal Range

In a severe mitral stenosis with mitral valve area of 1 cm² or less hardly adequate flow can be maintained at rest without reaching pulmonary oedema levels Slightest exertion will necessitate higher flow consequently the pressure in the pulmonary capillaries will exceed the pulmonary oedema level and pulmonary oedema will result

With the mitral valve area of 0 4 cm² flow and therefore the cardiac *output are diminished at rest In order to maintain even this diminished* mitral valvular flow the pulmonary capillary pressure has to work at the border line of the pulmonary oedema and the pulmonary congestion may exist even at rest

Formation of pulmonary oedema —

The pulmonary oedema is precipitated by any one or more of the following factors —

1 Tachycardia—due to effort infection etc by reducing the diastolic filling period and raising the cardiac output
2 Hypervolemia—by raising the cardiac output after infusions etc
3 Probably neurogenic factors—emotion excitements etc
4 Probably chemical—by increasing the pulmonary capillary permeability anoxia etc

Of all the cases with acquired heart disease presented here a few of the PW pressure obtained were observed at rest to be above 35mm Hg i e on the threshhold of pulmonary oedema In these few cases the PW pressures during exercise went up considerably higher than the plasma colloid osmotic pressure without any actual production of the pulmonary oedema The highest PWm pressure at rest observed in the present series is 37mm Hg In one patient who went in chill during the cardiac catheterization showed PWm as high as 44 mm Hg Even after levelling of the arbitrary 0 point in relation to the tip of the catheter leaving the margin for the errors of the technique of possible defective respiratory variations etc one has to explain these inordinately high PWm pressures The following factors have been ascribed to account for the prevention of the pulmonary oedema in the presence of so high PWm pressures

1 One of the most important observations made in the pathology of the lungs in long standing mitral stenosis is the thickening of the capillary basement membrane It is very logical to think that the thickened capillary basement membrane is less permeable and to that extent prevents the transudation This can explain the slowing down of the oedema formation temporarily

but highly elevated PWm pressures at rest for a long time the absence of oedema is rather hard to be explained by this circum stance

2 Distensibility or the elasticity of the capillary walls and the enormous number of the pulmonary capillaries —

Under normal conditions when an individual is subjected to exercise the required increase in the amount of pulmonary flow without significant rise in pressure is accomplished both by the increase in the distensibility of the capillary walls and the opening of the reserved pulmonary capillaries

3 Broncho motor tone [31]

Rodbard's concept of broncho motor tone involves an extrinsic mechanism of the rise in the intra alveolar pressure to balance the increased pulmonary capillary pressure This factor of extra-capillary regulation of the pulmonary blood flow by counterbalancing the rise in pulmonary capillary pressure appears to be operating through the changes in the bronchiolar lumen

It is shown by anatomists that the bronchial and the bron chiolar walls have a very efficient and capable muscular element which is controlled by the autonomic nerve supply The diminished tension in the alveoli during the inspiration and the total blood volume in the lungs rise During expiration bronchioles have a tendency to constrict and therefore some air is trapped in the alveoli during expiration thereby raising the intra alveolar tension The increase in the intra alveolar pressure behaves as a counteracting force against the rise in the pulmonary capillaries pressure and prevents any transudate across the capillaries

Whenever the patients of mitral stenosis are subjected to exercise their high left atrial and PW pressure are suddenly elevated Their actual pulmonary capillary pressure required to pump against the elevated left atrial pressure will exceed the osmotic pull of plasma protein without much production of the pulmonary congestion The situation like this lends support to the hypothesis of the regulation of pulmonary capillary flow through some extra capillary sources However the relationship between the degree of the rise in the intra alveolar pressures and its effectiveness on the pressure in the pulmonary capillaries is not linear The Rodbard's concept appears to be very interesting but it is still highly hypothetical and certainly demands much more documentation

4 Physiological increase in pulmonary vascular resistances

One of the most important protective mechanism for the prevention of pulmonary oedema is the increased pulmonary vascular resistence due to spasm of the pulmonary arterioles The exact mechanism of the spasm of pulmonary arterioles is not definitely understood However reflex neurological or chemical factors like anoxia may be working together Whatever the mechanism may be the higher the pulmonary vascular resistance less the chances of pulmonary oedema

5 Shunt mechanism

There are extra pulmonary bronchial veins (pleuro hilar veins)[17] draining into azygos hemi azygos and inter costal veins communicating freely with pulmonary veins These extra pulmonary veins are dilated and varicose in mitral stenosis[18]

It therefore provides a proof that the pulmonary venous circulation possesses a safety valve in these extra pulmonary bronchial veins which can effectively lower the pulmonary capillary pressure at the cost of cardiac output Whenever shunt mechanism is functioning effectively the right ventricle is enlarged out of proportion with the physiological data obtained by routine cardiac catheterization

It is interesting here to point out that the three manifestations of the pulmonary congestion namely orthopnoea paroxysmal nocturnal dyspnoea and the acute pulmonary oedema occur in the course of the disease in the order mentioned but the improvement in the natural course of the disease is in the reverse order i e first to disappear is the attacks of pul monary oedema than paroxysmal dyspnoea and orthopnoea persists till last in the disease of the severest form

FATIGUE AND DYSPNOEA IN PREDOMINANT MITRAL REGURGITATION

The fatigue is a more common complaint of patients with predominant mitral regurgitation Depending upon the degree of regurgitation the group of patients primarily complain of easy exertional fatigue tiredness and weakness These patients do have moderate dyspnoea and some of them paroxysmal nocturnal dyspnoea The development of fatigue and dyspnoea in predominant mitral regurgitation can be divided into three distinct stages —

1 Asymptomatic

The left ventricle being competent ejects the normal aortic output in addition to the blood lost in mitral regurgitation Such

mild regurgitatation does not lead to pulmonary hypertension of any consequence

2 Fatigue as a chief complaint

The left ventricle while losing significant amount of blood of regurgitation is beginning to fail to meet with the body demand for the cardiac output. The usual apportioning in a predominant regurgitation leading to the fatigue as a chief complaint is 50% of the left ventricular output through the mitral valve leak and the remaining 50% being delivered to the body Here the primary physiological defect is low cardiac output and the pulmonary hypertension is of the secondary importance However in many of the cases this stage of fatigue as a chief complaint may be totally absent

3 Fatigue and dyspnoea, both as chief complaints

The left ventricle is finally in the state of failure and the left ventricular diastolic pressure is considerably high This advanced left ventricular failure will give rise to severe pulmonary hypertension in addition to the low cardiac output Thus during this stage the symptoms of left ventricular failure in both the directions will arise In the situation when left ventricular early diastolic rapid dilatation causes the inter ventricular septal bulging towards the cavity of the right ventricle the right ventricular filling is naturally interfered with In such cases with inadequate right ventricular function production of severe pulmonary hypertension cannot occur The symptoms of pulmonary congestion in a situation like this are the result of low cardiac output causing poor renal flow and elevated venous pressure due to hypervolemia and Bernheim effect Thus symptoms of pulmonary congestion in the predominant mitral insufficiency are the result of left ventricular overloading while in mitral stenosis overfunction of the right ventricle is the causative factor

HAEMOPTYSIS

This symptom was twice as frequently observed in cases of pure or predominant stenosis as in mitral insufficiency and occurs only after fairly advanced degree of pulmonary hypertension

The haemoptysis can occur from any one of the following sources —

1 Pulmonary capillaries—congestion
2 Rupture of small intra pulmonary bronchial veins[19]

3 Infective bronchitis with oedematous bronchial mucosa
4 Pulmonary infarction

The heamoptysis caused by 1, 2 and 3 above presumably results from the high pulmonary venous or capillary pressures with an occassional increase in the pulmonary flow The pulmonary venous or capillary walls are not significantly thickened and the pulmonary arteriolar resistance is not significantly elevated The haemoptysis occurs relatively early in the course of the disease and subsides gradually in the natural course of the disease when the protective mechanism for the pulmonory capillaries and veins is developed well Thus a patient with marked pulmonary hypertension is generally free from this type of haemoptysis[23] The haemoptysis from the pulmonary infarct is generally because of an embolus dislodged from phlebo thrombosis in the lower extremities

The history of haemoptysis was obtained in the following type of patients in the present series —

1 The mitral valve area was between 0 5 to 1.25 cm² which gave rise to fairly severe degree of pulmonary hypertension as shown in figure 75

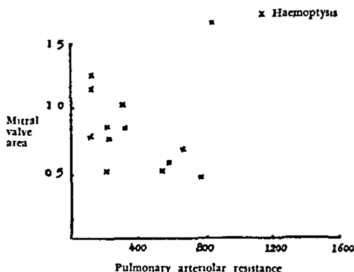


Figure 75
Predominant mitral stenosis
Graph showing the response of the pulmonary arteriolar resistance to the mitral valve area in relation to the history of haemoptysis
Case Nos 101 103 104 107 108 110 111 112 114 202 408 410

2 The pulmonary arteriolar resistence was not very highly elevated The figure 75 shows that the maximum observed in the presented group is 760

3. The right ventricular pressure work was increased showing that the right ventricle was competent enough to eject more blood than can pass through the mitral opening leading to the congestion of pulmonary capillaries

4. The cardiac output of these patients was only moderately diminished

Symptoms Of Right Sided Failure (Hepatomegaly peripheral oedema etc.)

Theory predicts that the progressively increasing pulmonary arteriolar resistances in the course of mitral disease increase the right ventricular work load In cases of mitral disease the elevated pulmonary arteriolar resistence is conventionally considered to play a significant role in the causation of the right ventricular failure

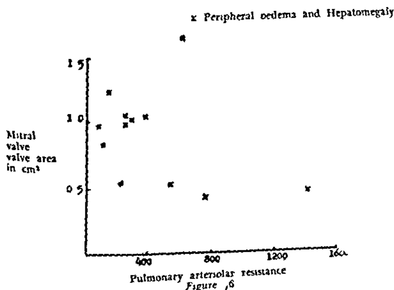


Figure 6
Predominant mitral stenosis
Graph showing the response of the pulmonary arteriolar resistance to the mitral valve area in relation to the peripheral oedema and hepatomegaly
Case Nos 103 106 108 109 111 112 113 114 201 206 416

However in the present series the right sided failure is noted with the relatively low pulmonary arteriolar resistences in the majority of cases It was observed that the patients having the right sided failure with relatively low pulmonary arteriolar resistence showed some evidence of myocardial disease in as much as their cardiac output was markedly diminished and their pulmonary hypertension was very mild

In some cases with extreme pulmonary hypertension or advanced myocardial disease the tricuspid insufficiency develops on the functional

basis This is an additional burden on the right ventricle and considerably further reduces the cardiac output In these cases hepatomegaly is disproportionately more marked than the peripheral oedema

Generally the symptoms of right sided failure appear gradually after a period of pulmonary congestion Dyspnoea and orthopnoea may be alleviated due to the reduction in right ventricular output Symptoms and signs due to right sided failure appear to be due to the congestion of systemic veins such as engorgement of cervical and other superficial *veins hepatomegaly oedema and transudates in serous cavities*

Emboli The thrombi may occur in both atria first in the large left atrium and its appendage and later with the development of the right sided failure right atrium is involved Fragments of these thrombi may get dislodged and produce embolic phenomena in the cerebral peripheral or visceral arteries in order of frequency The emboli can be multiple or recurrent [5,27] The systemic emboli are slightly more frequent in mitral stenosis than in mitral insufficiency presumably because of greater stasis in left atrium in mitral stenosis [22,27] The incidence of embolisation has no correlation with the size of left atrium or its appendage the elevation in pulmonary vascular resistance or the diminution in the mitral valve area The onset of atrial fibrillation is dangerous and may precipitate systemic emboli

Cough It is usually non productive in pure mitral stenosis and generally due to pulmonary congestion Rarely it may be due to the pressure of a large left auricle on the bronchus The sputum contains heart failure cells

Hoarseness of voice The change in voice may be due to compression of the left recurrent laryngeal nerve partly by the enlarged left auricle and partly by the enlarged tracheo bronchial lymph nodes and perhaps by the enlarged pulmonary[7,11,19] artery also

Weakness The weakness exhaustion and tiredness as described by the patient may be due to excessive work of respiration and inadequate cardiac output

Anginal syndrome The anginal pain is more common in severe mitral stenosis with high pulmonary vascular resisatnce than in mitral insufficiency After successful valvotomy the pain often disappears In these cases coronary arteries do not show any lesion at autopsy It is therefore believed that the pain may be attributed to diminished coronary filling due to diminished cardiac output[27]

Cyanosis This is not a major feature of the disease It may appear late in the course of the disease and is due to hyperplastic sclerosis of the

pulmonary arteriolar walls, thickening of the basement membrane and vascular endothelium and the stagnation of blood in the venules The gaseous interchange is markedly affected because of the thickened partition of the alveolar tissue separating blood from the alveolar air

The cyanosis may appear early if pulmonary oedema develops During the attack of pulmonary oedema arterial oxygen saturation can fall significantly low (even 50%) when anoxia can alter pulmonary capillary permeability

Peripheral pulse In uncomplicated cases of both mitral insufficiency and mitral stenosis the peripheral pulse is small in volume because of diminished cardiac output but the peripheral pulse in mitral insufficiency may be waterhammer type because of systolic leak from the arterial system while that in mitral stenosis may be low in tension because of diminished systolic pressure in the left ventricle and arteries

Arrhythmia Auricular fibrillation is the most important cardiac irregularity noticed The exact mechanism of the production of auricular fibrillation in mitral disease is not understood but the rheumatic activity in the myocardium may be responsible though the study of the atrial biopsy from cases undergoing valvotomy does not confirm this The unusual dilatation of the left or right auricle in cases of mitral stenosis with auricular fibrillation could be the result rather than cause of arrhythmia It is interesting to note that the patients with auricular fibrillation are less likely to run into pulmonary oedema easily because the cardiac output is reduced with the onset of the fibrillation

Jugular venous pulse The character of jugular venous pulse in mitral insufficiency and mitral stenosis is a function of generalized elevation in the right sided venous pressure However the v wave in mitral insufficiency is generallly more conspicuous than in mitral stenosis because of inadequate right ventricular filling due to pronounced Bernheim effect in mitral insufficiency

Apex Beat With an enlarged left ventricle in mitral insufficiency the apex beat is shifted to the left and it is somewhat sustained With adynamic left ventricle in mitral stenosis the apex beat is hardly palpable However in mitral stenosis the enlarged right ventricle shows its pulsation from the left para sternal line to the epigastrium

Ist Apical heart sound The first heart sound in mitral insufficiency is soft or normal while a loud and sharp apical first sound is one of the early signs of pure mitral stenosis In mitral stenosis elevated trans mitral pressure gradient keeps the mitral cusps widely opened until late diastole These valve cusps close very forcefully during left ventricular

isometric contraction period because of fibrotic changes in the cusps and chordae tendinae It is this forceful closure of the cusps that is partly responsible for accentuated 1st round Forceful contraction of the left ventricle also is partly responsible for production of sharp and booming 1st sound of pure mitral stenosis

In atrial fibrillation the atrio ventricular pressure gradient late in diastole will be smaller when the preceding diastole is long Therefore the intensity of the first sound which depends upon the late diastolic atrio-ventricular pressure gradient will vary inversely with the length of the preceding diastole [20]

Second Pulmonary Heart Sound The accentuation of P_2 is partly the result of an elevated pressure in the pulmonary artery and partly of rotation of pulmonary artery and conus bringing them closer to the chest wall The reduplication of P_2 occassionally heard is probably due to the change in relative pressures in the pulmonary artery and the aorta leading to asynchronous closure of the semilunar valves The degree of accentuation of P_2 in the present series was not expected to have any close correlation with the severity of pulmonary hypertension

Presystolic Mitral Murmur The presystolic murmur is caused by the left atrial systolic contraction squeezing the last portion of blood through the stenosed mitral valve This is a very early sign of mitral stenosis even if the left atrio ventricular pressure gradient is only 5 to 10 mms Hg provided there is a normal rhythm However it could be masked later in the course of the disease when the grossly enlarged right ventricle displaces the left ventricle and the mitral valve posteriorly The presence of presystolic murmur can rule out significant degree of mitral insufficiency The crescendo effect described as typical of mitral stenosis is really an auditory illusion because of the murmur abruptly ending into short and sharp 1st sound

Early and Mid Diastolic Murmur This occurs in a fairly severe degree of mitral stenosis unaffected by auricular fibrillation The onset of this murmur is timed with the beginning of rapid diastolic filling i e the descending limb of v wave The length of the murmur can indicate the severity of stenosis In mild mitral stenosis the left atrio-ventricular pressure gradient disappears relatively early in diastole while in severe mitral stenosis the left atrio-ventricular pressure gradient is carried through the entire diastole

A mitral murmur in early diastole may be present in pure mitral insufficiency because of the large volume of blood flowing across the mitral valve in early diastole with an elevated trans valvular pressure gradient

Systolic Murmur The systolic murmur in mitral insufficiency replaces the 1st sound and continues into the 2nd sound The mitral leak commences during the isometric contraction period because of the left ventriculo atrial pressure gradient and continues into the proto diastole because of the continued ventriculo atrial pressure gradient until the closure of aortic valve Thus the apical pan systolic murmur is characteristic of mitral insufficiency However an apical systolic murmur may be present in pure mitral stenosis because of calcification of the mitral cusp [11]

Thus an early diastolic murmur can be present in pure mitral insufficiency and a systolic murmur can exist in pure mitral stenosis The intensity of the diastolic or the systolic mitral murmur partly depends upon the amount and the rapidity of the blood flow across the mitral valve during the left ventricular diastole or the systole respectively The loudness of the murmur does not depend upon the severity of the mitral valve disease only With a very low cardiac output the diastolic murmur may be very faint despite the severest mitral stenosis Similarly with a very low cardiac output and the big mitral valve regurgiant area the systolic murmur fades away,

Opening Snap of the Mitral Valve This was described by Duroziez as early as 1862 [*]

At the end of isometric relaxation the maximally elevated left atrioventricular pressure gradient flings open the aortic cusp of the mitral valve into the ventricular cavity causing the opening snap It is short sharp and clicking in character usually heard best in anatomical position of the mitral valve i e the 3rd and 4th intercostal space The sound is produced by sudden curtailment of the opening movement of the stenosed mitral valve as the blood from the auricle to the ventricle begins to flow in early diastole

Wherever the mitral cusps are supple the opening snap should be heard However the opening snap can be masked by enlarged right ventricle or associated aortic incompetence The absence of the opening snap in mitral insufficiency is probably due to the fibrous ankylosis of the aortic cusp of the mitral valve

The opening snap occurs about 0 10 second after the 2nd sound and just before the beginning of the diastolic murmur This corresponds with the apex of the v wave of the left atrial tracing.[11]

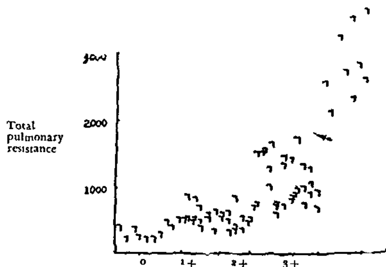
Next to the characteristic murmur the opening snap has a great practical value in the diagnosis of mitral stenosis being present in more than 50% of cases of pure mitral stenosis and many a time it is easily heard

It is to be differentiated from the proto diastolic gallop rhythm the reduplication of the second sound and the normal third heart sound

Graham steel murmur The functional diastolic murmur in the pulmonary area could be heard only after a severe pulmonary hypertension is developed

Right Ventricular Enlargement On fluoroscopy and electro cardiography

The fluoroscopic enlargement of the right ventricle was graded as plus minus one plus two plus and three plus



Fluoroscopic right ventricular hypertrophy graded from plus minus to 3 plus

Figure 77

Graph showing the relationship of the total pulmonary resistance with the fluoroscopic hypertrophy of the right ventricle in both mitral stenosis and regurgitation

Case Nos	101	102	103	104	105	106	107	108	109	110	111	112	113	114	118
	126	12	128	129	131	132	133	135	151	152	153	154	155	201	203
	205	206	226	227	229	230	251	401	402	403	404	405	406	407	408
	409	410	412	413	414	415	416	417	418	419	420	421	422	423	424
	451	452	453	454	455	456	457	458	459	460	461	462			

Fluoroscopic right ventricular hypertrophy in four grades— 0 1 2 3

The figure 77 shows the significant correlation of the right ventricular enlargement with the total pulmonary resistance The cases plotted in the figure include those having predominant mitral stenosis as well as predominant mitral regurgitation

Right ventricular enlargement	Total pulmonary resistance
Plus minus	240 to 540
One plus	340 to 880
Two-plus	630 to 1770
Three plus	2130 to 3640

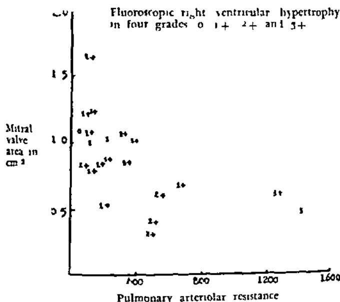


Figure 78

Graph showing the response of the pulmonary arteriolar resistance to the mitral valve area in relation to the right ventricular hypertrophy fluoroscopically in mitral stenosis

Case Nos 101 102 104 106 107 108 110 111 112 113 118 201 203 05 206

Furthermore in cases of pure mitral stenosis the right ventricular enlargement became more marked as the mitral valve area decreased and the pulmonary arteriolar resistance increased

The electro cardiographic evidence of the right ventricular enlargement was noted in a wider range of pulmonary hypertension There was no working correlation observed between the degree of the pulmonary hypertension and any of the criteria for the right ventricular enlargement in the standard leads augmented leads and unipolar leads of the electro cardiograph This observation is not quite consistent with the findings in Paul Wood's series[24]

Left Atrial Enlargement (by fluoroscopy and electro cardiography)

The fluoroscopic enlargement of the left atrium was graded plus minus one plus two plus and three plus

As shown in the figure 79 in cases of pure mitral stenosis there is a general tendency of increasing enlargement of the left atrium as the mitral resistance increased Neveretheless the onset of change in rhythm plays perhaps a significant role in the enlargement of the left atrium

The systolic pulsation of the left atrium is not considered a consistent sign of the mitral regurgitation as could be determined from PW tracing and/or cardiotomy

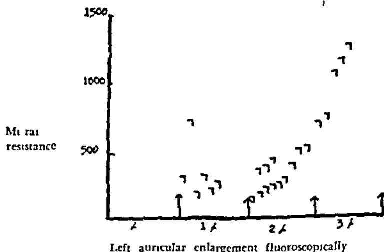


Figure 79

Graph showing the relationship of the mitral resistance (difference between the total pulmonary resistance and pulmonary arteriolar resistance) with the enlargement of the left auricle in mitral stenosis

Case Nos 101 102 103 104 106 107 108 109 110 111 112 113 114 118 201 205 403 405 408 409 410 415 417 418

Roentgen Enlargement of the Pulmonary Artery Attempts have been made to find a significant correlation between the size of the pulmonary artery segment and the degree of pulmonary hypertension However inconsistencies are found quite frequently and therefore such a correlation certainly invites skepticism[26] although marked dilatation of the pulmonary artery suggests elevated pulmonary resistance and therefore severe mitral stenosis

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CHAPTER V

NATURAL LINES OF DEFENCE FOR THE SYMPTOMS OF THE PULMONARY CONGESTION IN MITRAL STENOSIS

Human body is provided by nature with the means for its protection whenever there is anything wrong with it Thus with the progressive degree of mitral obstruction there are four different line of defense coming into action This natural course of reactions of the protective body mechanism comes into play immediately as the mitral valve area decreases below 2 5 cm² If the entire circulatory system is looked upon as a series of chambers valves and pipes the obstruction to the blood flow at any one point will have similar effect as would happen when a dam is constructed in the path of a flowing river Thus mitral dam will produce stagnation of blood proximal to the dam i e in the pulmonary circulation

The first line of defense consists of four different changes which start working to maintain an adequate blood flow across the mitral valve

1 The pressure in the pulmonary capillaries rises in order to maintain adequate cardiac output The high pulmonary capillary pressure may produce more pulmonary capillary transudate resulting into dyspnoea

2 Left atrial activity is magnified i e left atrial systole is powerful leading to the left auricular enlargement fluoroscopically

3 Diastolic filling period is increased from 37 to even 42 and therefore systolic ejection period is decreased contributing to the production of the well known short first sound at the apex The sharpness of the 1st sound at the apex can be explained by the sudden rushing back of the mitral valve cusps into the auricle due to sudden rise of pressure at the beginning of the left ventricular systole

4 Opening of the new pulmonary capillary channels giving the effect of pulmonary congestion on screening

As this first line of defense is reaching its optimum the second line of defense is the reduction in cardiac output This usually happens when the mitral valve area is 1 cm With the reduced cardiac output the total turnover of the blood from the right ventricle to the left ventricle via the pulmonary capillaries is also reduced This may contribute towards the prevention of pulmonary oedema or congestion As the cardiac output is diminished the symptom of fatigue appears and the

work capacity further diminishes Although the first and second line of defense occur simultaneously the second is a less sensitive index and reacts second to the abnormal situation With the decreased cardiac output the intensity of the diastolic murmur may also diminish The development of auricular fibrillation at this stage can abolish the presystolic murmur and may minimize the diastolic murmur

The third line of defense for preventing the pulmonary oedema is by increasing the pulmonary vascular resistance physiologically and pathologically due to the spasms and thickening of the pulmonary arteriolar and capillary walls The pathological changes noted are intimal thickening medial hypertrophy narrowing of the lumen scarring thickening of the capillary basement membrane and transition to the cuboid epithelium All these changes lead to the narrowing of *the pulmonary vascular bed resulting in increase in the pulmonary resistance* At this stage it is interesting to note that with the thickened pulmonary capillary walls the transudate across the walls is less and therefore less pulmonary congestion This is clinically reflected through the alleviation in the symptoms of the pulmonary congestion like dyspnoea If the dyspnoea before was 3 plus with the advance of the disease it may become 1 plus or 2 plus Such apparent improvement is the natural course of the disease but sometimes it is erroneously attributed to the therapeutic measures such as digitalis Thus with such capillary changes right ventricular pressure head by the time it is transmitted through the *pulmonary capillaries is proportionally reduced the pulmonary vascular* bed diminishes and the right ventricular pressure work increases

Finally the maintenance of the high pressure in the pulmonary circuit for a long time requires very competent myocardium but ultimately the right ventricular myocardial capacity is also limited particularly because the myocardium has undergone acute rheumatic myocarditis at one time or the other Whenever the myocardium is considerably diseased the right ventricle begins to fail much earlier without producing any significant pulmonary hypertension The onset of the right ventricular failure is not really a defence on the part of the body but it is merely a matter of shifting the congestion from the pulmonary circulation to the peripheral circulation

CH[illegible]

MITRAL STENOSIS VERSUS MITRAL REGURGITATION POSSIBILITY OF EXACT DIAGNOSIS

Clinicians have failed to discover any one particular criterion pathognomonic of advanced mitral regurgitation However there are some findings in favour of marked mitral regurgitation and some in favour of predominant mitral stenosis In order to evaluate a case properly all the available data must be weighed and considered according to their respective merits A clinician can be reasonably certain that in cases having typical findings of mitral stenosis such as apical diastolic murmur of grade 2 or more sharp first apical sound opening snap of the mitral valve fluoroscopic enlargement of the left auricle etc there can be no mitral regurgitation of a degree contra indicating valvuloplasty On the other hand with an apical systolic murmur and left ventricular enlargement fluoroscopically and electro-cardiographically (when other causes of the left ventricular enlargement are excluded) one can suspect the presence of only significant mitral regurgitation It is common experience that mild to moderate mitral regurgitation does not preclude benefits from mitral valvuloplasty for stenosis

Nevertheless exact diagnosis of the degree of mitral stenosis or mitral regurgitation has always puzzled the best of clinicians Methods like phonocardiography auricular oesophagograms and even auricular border electro kymorgrams have not been of any definite diagnostic value The calculation of mitral valve stenotic area and the mitral valve regurgitant area in double mitral lesions have neither been of much practical help

In the present series wherever necessary PW pressure tracing through the cardiac catheter or LA tracing obtained trans bronchially were considered for the pre operative evaluation of the degree of stenosis or regurgitation The height of v wave was carefully measured The peak of v wave 7mm Hg higher than the peak of c wave was considered as indicative of predominant regurgitation In cases of auricular fibrillation an average of the heights of c and v waves in 20 cycles was calculated

A clinician will be rewarded if he finds a reliable index in the respective heights of c and v waves of PW or LA pressure tracings for the exact diagnosis of the degree of mitral regurgitation. We have not always been able to solve our problems of assessing double mitral lesions satisfactorily but the help of PW or LA pressure tracing has been quite valuable (Refer to figures 80 81 and 82) However it is fully realized that occas



Figure 80

Trans—bronchial pressure tracing in left atrium in mitral stenosis Measurements of left atrial pressure *obtained by this method compare favourably with the pressure* obtained at operation and also with the levels and form of the tracing of pulmonary wedge pressure obtained by catheterization

Each vertical line is 0 2 sec apart

The speed of the paper is increased in the latter part

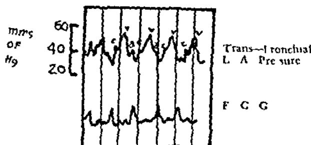


Figure 81

Trans—bronchial left atrial pressure tracing alongwith E C G in a patient with regular sinus rhythm and markedly prominent mitral insufficiency The height of wave is greater than that of c wave by about 12 mms Hg

Each vertical line is 0 sec apart

sionally in case of tight mitral stenosis the influx of small amounts of blood into a left auricle which is already distended beyond its capacity to expand readily may lead to a very high v wave in the absence of mitral insufficiency Therefore while the presence of v wave adds a positive point in favour of predominant mitral insufficiency its absence does not exclude mitral insufficiency In such cases Paul Wood and his associates have suggested that the ratio of the slope of the decline of the

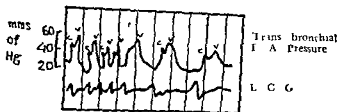


Figure 82

Trans-bronchial left atrial pressure tracing in a patient with auricular fibrillation and markedly predominant mitral insufficiency The average height of v wave is greater than that c wave by about 12 mm Hg
Each vertical line is 0 [illegible] sec apart

v wave (Ry/V' ratio) may help to differentiate the v wave elevation due to mitral insufficiency from that due to mitral stenosis

In mitral stenosis the rate of flow of blood across the mitral valve during diastolic filling period is slow and therefore the atrial emptying or the rate of atrial pressure fall is relatively gradual The trans mitral pressure gradient is demonstrable throughout the entire diastole Thus the obstruction to the mitral valve flow slow down the rate of descent of v wave Contrary to this in mitral insufficiency *trans mitral flow is rapid and therefore the pressure gradient disappears early in diastole* The rate of descent of v wave is rapid

The degree of obstruction to the forward flow is expressed by Owen and Wood[2] as Ry/V' ratio where Ry is the descent of v wave in mm Hg per second and v is the height of v wave in mm Hg After careful analysis of a number of cases Owen and Wood[2] have suggested that in mitral stenosis the Rv/v ratio ranges between 0 6 and 1 6 In mitral insufficiency left ventricular failure or chronic constrictive pericarditis the ratio lies between 2 and 6 The value between 1 6 and 2 indicated borderline cases The value of Ry/v ratio to distinguish predominant mitral stenosis from predominant mitral insufficiency has now been under observation

Others[1] have suggested that significantly high v wave above the mean L A pressure of 20 mm Hg or more is a good indication for predominant mitral regurgitation

However there are always a group of patients who have significant degree of both stenosis and regurgitation Besides associated deformity of aortic valve can also confuse the picture and one cannot properly evaluate mitral diseases under these circumstances It is in such cases that left heart catheterization would be mandatory for proper evaluation of mitral disease and associated aortic deformity

Simultaneous pressure tracings from left atrium left ventricle and aorta can elucidate the exact happenings at the mitral and aortic valve

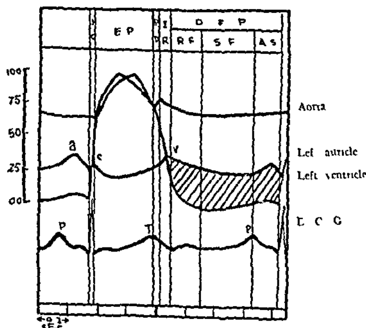


Figure 83

Simultaneous left auricular left ventricular and aortic pressure tracing (drawn theoretically) The shaded area indicates the elevated atrio—ventricular pressure gradient during ventricular diastole acros the stenosed mitral valve The slow descent of v wave also favours the pre ence of mitral stenosis

(Abbreviations are same as in Figure 1)

Figure 83 shows the typical left sided pressure curves in a case of pure mitral stenosis In normal persons left atrial mean pressure is around 4 to 8 mm and during ventricular diastole the required atrio-ventricular pressure gradient for necessary atrio-ventricular flow is hardly 1 or 2 mm Hg. while in mitral stenosis left atrial mean is elevated even up to 28 to 30 mm Hg in order to raise the atrio ventricular pressure gradient necessary for the required atrioventricular flow Thus the high atrio ventricular pressure gradient during ventricular diastole and a very slow descent of v wave speak for narrow mitral orifice

Figure 84 shows the typical left sided pressure curves in a case of severe mitral insufficiency There is no atrio ventricular pressure gradient during ventricular diastole v wave is tall and descent of v wave is very rapid Tall v wave indicated ventricular systolic regurgitation through the incompetent mitral valve The absence of atrio ventricular pressure gradient and rapid descent of v wave speak for widely opened mitral orifice during diastole The elevated left ventricular diastolic pressure shows limited accommodation of blood in the chamber

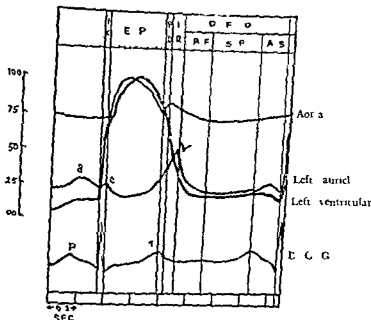


Figure 84

Simultaneous left auricular left ventricular and aortic pressure tracing (drawn theoretically) During ventricular systole the regurgitation of blood into the left auricle through the incompetent mitral valve causes a well known large V wave of the left atrial pulse The absence of abnormal atrio-ventricular pressure gradient during the ventricular diastole and the rapid descent of V wave excludes any significant degree of mitral stenosis

(Abbreviations are same as in Figure 1)

The application of dye dilution curves has also been of diagnostic value in cases of severe mitral insufficiency In these cases there is distortion of dye dilution curves recorded from a peripheral artery or left atrium directly irrespective of the site of injection The details of the technique are outside the scope of this presentation

The very fact that different groups are using different criteria to distinguish predominant mitral insufficiency from mitral stenosis suggests that better methods of such differentiation in doubtful cases are still needed

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CHAPTER VII

ROLE OF CARDIAC CATHETERIZATION IN SELECTING CASES FOR MITRAL VALVOTOMY

Pulmonary hypertension

The understanding of rheumatic fever is that both the myocardium and the valvular tissues are affected by it The symptoms of the disease are either mainly due to the insufficiency of the myocardium or the mechanical block at the mitral level What is accomplished by valvotomy is the correction of the mechanical block i e the valvular defect only Before submitting the patient therefore to surgery one has to make sure that the symptoms are predominantly the result of the mechanical mitral block The significant pulmonary hypertension indicates the predominant mechanical block and sufficient right ventricular myocardium Smaller degree of pulmonary hypertension in comparison with the severity of symptoms indicates predominant myocardial disease and inadequate right ventricular myocardium Therefore fair degree of pulmonary hypertension is necessary to expect a good result from valvotomy

To be considered for surgery the patient should have the following

1 Left atrial or pulmonary arterial wedge mean pressure should be over 12 mm Hg

2 Pulmonary arterial mean pressure should be at least 40 mm Hg to give pulmonary arterio venous gradient of about 28 mm Hg

3 Right ventricular systolic pressure should be nearer to 55 mm Hg and diastolic less than 6 mm Hg.

Cardiac output

The cardiac output should be close to 3 liters at rest and be raised or at least not fall during exercise Low cardiac output indicates high degree of obstruction or insufficiency of the myocardium

Degree of mitral obstruction

If Gorlins hydraulic formula is used mitral valve area should be around 1 plus minus 0 5 cm² If Owen & Woods Rv/V ratio is used it should be less than 1 6

Associated lesions

Significant mitral insufficiency

The height of v wave should not be more than 7 mm Hg higher than that of c wave Ry/V ratio should not be over 2.

Aortic lesions

Significant aortic lesions can be excluded by left heart catheterization but this rarely becomes necessary Aortic stenosis if severe enough may produce an anacrotic notch in the peripheral arterial tracing

Tricuspid lesions

Tricuspid stenosis or tricuspid insufficiency can easily be found from right atrial and the right ventricular tracing but these changes may not form absolute contra indication for mitral valvuloplasty

Chronic constrictive pericarditis

This also can be easily excluded from the right atrial and right ventricular pressure patterns and by watching the fluoroscopic course of the catheter

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CHAPTER VIII

CHANGING DYNAMICS FOLLOWING MITRAL VALVULOTOMY

Objective as well as subjective evaluation of the results of patients following mitral valvulotomy is absolutely necessary. In the present study an opportunity was taken to record the pulmonary arterial and left atrial pressures in the operating room immediately before and immediately after the mitral commisurotomy in over 40 cases. Only 3 were studied by re catheterization.

Important changes in the dynamics are as follows —

1. Pulmonary hypertension

   In the present series the changes in the pulmonary arterial tracing immediately following the fracture of the mitral valve were recorded simultaneously with the left auricular tracing. The drop in the pulmonary artery pressures immediately after valvulotomy was not *impressive*. The *adjustment* of pressures in the pulmonary circuit apparently takes some time after opening of the mitral valve. One of the reasons for this may be that the right ventricle working against the increased resistance continues to work with the *same force for some time*. Another reason is that the increased pulmonary arteriolar resistance which develops in the course of the disease still persists. Thus the pulmonary arterioles continues to act as an effective block against which the right ventricle has to work.

2. Left atrial or 'PW' pressures

   In all cases of severe mitral stenosis LA mean pressure dropped promptly and the PC' *pressure therefore* must have dropped[1,2,3,4,5,6,7,8,9,10,11,12,13,14]. However in many cases where significant mitral regurgitation existed before or after the opening of the mitral valve the drop in left atrial pressure was *less striking*. The significant drop in the left atrial pressure immediately following valvulotomy suggests that there has been significant decrease in the resistance to the flow through the *mitral valve*. However it should be *emphasized* that unless diastolic flow through the valve remains constant the changes in left atrial pressure or even LA—LV' diastolic pressure gradient are *of little* significance *since a fall in the diastolic* flow will also produce decreased LA—LV' pressure gradient.

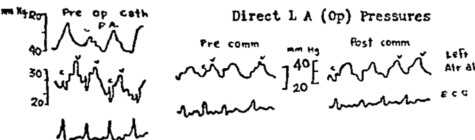


Figure 85

Figure showing the left atrial pressure tracing during catheterization (PW) immediately pre and post commissurotomy during surgery in a rheumatic cardiac patient with mitral stenosis and mitral insufficiency. The pulmonary artery wedge pressure shows a large V wave due to mitral insufficiency and the pulmonary artery pressure was greatly increased from 70/40 to 110/40. The v wave is tall indicating associated mitral insufficiency. At operation the predominantly stenotic mitral valve was fractured. The post commissurotomy curve shows increase in mitral insufficiency. At the same time the relief of the mitral stenosis caused a drop in pressure in the left atrium from 50/30 to 35/20 a very significant fall.

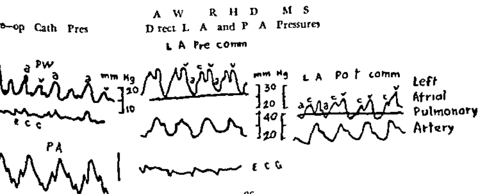


Figure 86

Figure showing the pulmonary artery and left atrial pressure tracing during catheterization (PW) pre and post commissurotomy. This shows greatly increased pulmonary arterial wedge and pulmonary artery pressures before operation and striking improvement with falls in the left atrial and pulmonary artery pressures after commissurotomy.

3 Mitral insufficiency

The diagnosis of mitral insufficiency was suspected mainly from the clinical picture and was substantiated by fluoroscopy and analysis of PW or LA pressure tracing. Most of the times this was confirmed by the surgeon's finger feeling a regurgitant jet before and after the fracture of the mitral valve. In some cases there was no insufficiency before or after the surgery, in some mitral insufficiency was marked following the fracture only, in others the degree of insufficiency was not accentuated after

surgery and in the rest mitral insufficiency was markedly accentuated after the surgery However it was apparent that in properly selected cases mitral valvuloplasty can relieve the stenosis without producing insufficiency of any consequence

4 Mitral valve stenotic area

In all the cases operated mitral valve stenotic area was adequately enlarged However in addition to the mechanical mitral block associated lesions in either the pulmonary vasculature or the myocardium can also interfere with the restoration of health after surgery Of the three equally important defects in cases of rheumatic heart disease with a mitral stenosis namely mechanical myocardial and vascular the surgeon s finger corrects only the mechanical one

5 Cardiac output

It is believed that a considerable time interval elapses before any significant rise in cardiac output really occurs This is partly due to the fact that reversible part of the pulmonary arteriolar resistances takes time to revert and partly because the atrophic left ventricular myocardium takes some time to adapt itself in order to receive and eject the increased amount of inflowing blood However the failure of the right ventricle to increase its output appears to be the main factor because if the left ventricular disability were playing any significant role it would immediately reflect in the elevation of PC pressures In some cases inevitably created mitral insufficiency during the fracture can account for the absence of expected rise in cardiac output

6 Pulmonary arteriolar resistance

It has been hypothesized that a part of elevated pulmonary arteriolar resistances pre operatively is due to the physiological vaso constriction of the pulmonary arterioles and the rest is due to the organic narrowing of the arterioles and capillaries[12] The dramatic decrease in pulmonary arteriolar resistances in 2 or 6 cases of Dexter et al[5] after surgery lends support to this fact However it is impossible to predict as to how much of the elevated pulmonary arteriolar resistances is reversible

7 Right ventricular function

This can be judged from the right ventricular diastolic filling pressure and the right ventricular pressure work In cases with predominant mitral mechanical block the general trend of the right ventricular function to revert to normal can be expected The cases with evidence of significant myocardial disease were carefully avoided for surgery

8 Ry/V' ratio

This ratio increases significantly after valvulotomy[1]

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Part III

CHAPTER I

INADEQUATE RIGHT VENTRICULAR LATE DIASTOLIC ACCOMMODATION

PERICARDIAL CONSTRICTION

Lowers in 1669 suggested the restricting effect of the constricting fibrous pericardium on the cardiac diastole. Two centuries later Chevers[3] first explained the mechanism of the chronic constrictive pericarditis. Pick (1896) after whom sometimes the disease is named—Pick's Disease[6] only stressed the part of hepatic cirrhosis. It is known only in the last century that the crux of pathologic physiology in chronic constrictive pericarditis lies primarily in the mechanical interference with the distension and the contraction during the ventricular diastole and systole respectively, especially the former.

However, the characteristic pressure patterns in the right auricle and the right ventricle were not recognised before the days of the right heart catheterization. Bloomfield *et al* first recognised and described the typical pressure patterns in constrictive pericarditis. This was confirmed by Wood *et al* and later by other workers[4]. Burwell and his associates stressed the clinico physiologic aspects. Hansen and his co-workers demonstrated partly reversible nature of the pressure pattern after the surgical resection of constricting pericardium. The derangement of various other haemodynamic data as obtained by cardiac catheterization was emphasized by Sawyer et *al*. Recently Harvey *et al* showed the variability of the haemodynamic data in chronic constrictive pericarditis from case to case depending upon the exact extent and the site of predominant cardiac involvement.

After the pre and post operative study of constrictive pericarditis it is now appreciated that the right atrial and ventricular pressure pattern although typical of chronic constrictive pericarditis does not always revert completely to normal despite adequate decortication. It has not been possible to exactly assess the role played by the pericardial restriction in producing the characteristic haemodynamic pattern.

Nevertheless the haemodynamic pattern is very characteristic of chronic constrictive pericarditis although it is not pathognomonic as was supposed initially by several workers. There are other diseases like intrinsic myocardial disease and certain congenital heart diseases[11] which do give rise to similar haemodynamic pattern. In order to distinguish

constrictive pericarditis from the other simulating diseases Yu et al[13] proposed that the ratio $\frac{\text{Right ventricular end diastolic pressure}}{\text{Right ventricular systolic pressure}}$ in constrictive pericarditis is always greater than 1/3rd. Later Wilson and his associates[14] modified the ratio $\frac{\text{Right ventricular end diastolic pressure}}{\text{Right ventricular systolic pressure}}$ should be elevated to at least 40% to be significant to show constrictive pericarditis.

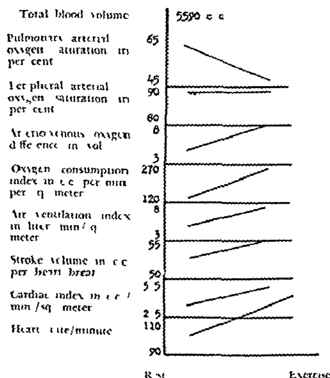


Figure 87

Graph showing the data obtained during the cardiac catheterization at rest and during exercise in a case of constrictive pericarditis proved at surgery

In a case of chronic constrictive pericarditis proved at surgery the typical pressure patterns and the haemodynamic data are shown in the Figure 87, 88 and 89.

The haemodynamic pattern shows the following characteristics —

Right ventricular tracing

1. Slightly elevated systolic pressure
2. Prominent early diastolic dip

Right ventricular work in Kgm /min / sq meter

1 00

0 40

Right ventricular end-diastolic pressure in mm Hg and in terms of % of right ventricular systolic pressure

30

50%

20

End diastolic as % of systolic

50%

End diastole

Right ventricular systolic and diastolic pressure in mm of Hg

60

Mean

10

systolic

diastolic

Pulmonary arterial systolic diastolic and mean pressure in mm of Hg

60

systolic

Mean

diastolic

20

Peripheral arterial systolic and diastolic pressure in mm of Hg

125

Mean

65

systolic

Diastolic

min 4 min 6 min

Rest Exercise Post Exercise

Figure 88

Graph showing the data obtained during the cardiac catheterization at rest and during exercise in a case of constrictive pericarditis proved at surgery

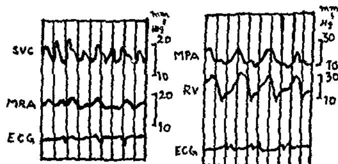


Figure 89

Pressure tracings in a case of constrictive pericarditis proved at surgery

SVC	Superior Vena Cava	MPA	Main Pulmonary Artery
MRA	Mid Right Atrium	RV	Right Ventricle
	ECG Electro-cardiogram		

3 Early diastolic dip does not reach O line

4 Rapid elevation of the diastolic pressure during the later 2/3rd of the diastolic period

5. Low ventricular pulse pressure
6. During tachycardia of exercise the high end-diastolic pressure could be graphically eliminated
7. The ratio
$$\frac{\text{R V end diastolic}}{\text{R V systolic}}$$ is greater than 40%

Right auricular tracing

1. Elevated right atrial mean pressure
2. V shaped excursion at the time of ventricular early diastolic dip
3. Greater atrial pulse pressure
4. c wave is almost absent or insignificant

Pulmonary arterial tracing

1. Slight elevation in systolic pressure
2. Moderate elevation in diastolic pressure
3. Relatively small pulse pressure

Pulmonary arterial wedge tracing

This showed more or less similar pattern as that in the right atrium provided that the mitral disease is excluded

Left atrial tracing

This can be obtained by a needle puncture from the opened chest at the operation for decortication or else trans bronchial left atriography[17] can be performed. These procedures were done in some cases in the present series and presented the same general characteristics as that in PW tracing

Cardiac output

Cardiac output is markedly diminished but the stroke volume is only moderately diminished

The physiologic defect in chronic constrictive pericarditis is essentially the inadequate ventricular late diastolic filling and to some extent the ventricular contraction may also be hampered. The limitation in diastolic filling is more or less same in both the ventricles

MECHANISM OF PRODUCTION OF THE HAEMODYNAMIC PATTERN

Ventricular pressure pattern

At the time of ventricular diastole the ventricle cannot be filled up completely because of the limitations in the distensibility imposed by the fibrous contracting pericardium. During ventricular systole the ventricle

is easily able to eject this much blood and therefore during early next diastole the ventricular content is at its minimum. This results into a very high pressure gradient across the atrio-ventricular valve. This constitutes early diastolic dip. The high pressure gradient fills up the ventricles quickly to its maximum capacity. There comes the limitation of the ventricular distension by the pericardial constriction which gives rise to late diastolic plateau.

Auricular pattern

The ventricular end-diastolic pressure is high and therefore the auricular mean pressure has got to be high in order to maintain the pressure gradient across the atrio-ventricular valve. As the ventricle fills up rapidly during early diastole the drop in the auricular tracing (fall of c wave) is coincident with the right early diastolic dip. When the ventricular diastolic pressure is high during the end-diastole the drop in the auricular pressure is not marked. Usually c wave is produced by the bulging of the valvular cusps in the auricles during isometric contraction. Here because of the limitation of the constricting pericardium the ventricular contractility is markedly restricted and therefore hardly any forceful bulging of the valvular cusps occurs. It is this abolition of c wave which gives rise to 'M' of 'W' shaped auricular pattern.

Pulmonary hypertension

The degree of pulmonary hypertension here indicates the extent of left heart pericardial restriction provided mitral disease or left ventricular failure is excluded. The pulmonary arterial pulse pressure is smaller because both ventricles are affected more or less to a similar degree.

Thus it is derived that the change of pressure pattern in the ventricles is primary and that in the auricles and pulmonary circuit is secondary.

HAEMODYNAMIC BASIS FOR THE SYMPTOMS AND SIGNS

Predominance of systemic congestion and oedema

As the limits of distensibility produced by the constricting pericardium is generally similar for both the ventricles the elevation of venous pressure in greater and lesser circulation is also of a similar degree[3].

The consistant observation by all the investigators in the field is that the normal difference in pressures in the venous channels right heart and pulmonary circuit during the ventricular diastole is minimized. Normally the hydrostatic pressure in the systemic capillaries is just in balance with the plasma osmotic pressure while the pulmonary capillaries which are designed normally to work at a much lower hydrostatic pressure is provided with a greater margin of safety before it exceeds the osmotic plasma

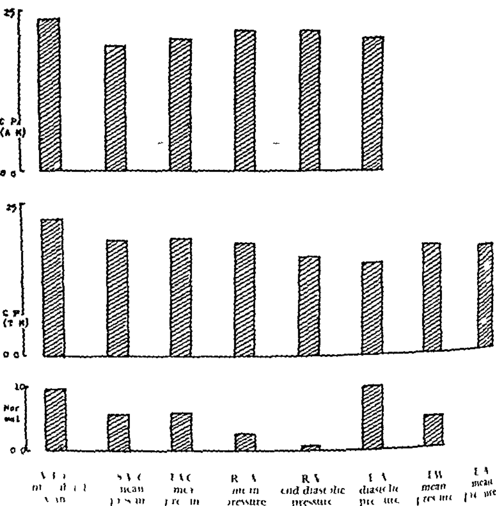


Figure 30

Figure showing the mean pressures at different places in the right side of the circulation in case of constrictive pericarditis (C P) as compared to normal

V P	Venous Pressure	R V	Right Ventricle
S V C	Superior Vena Cava	P A	Pulmonary Artery
I V C	Inferior Vena Cava	P W	Pulmonary Wedge
R A	Right Atrium	L A	Left Atrium

pressure levels Now in cases of constrictive pericarditis the pressure rise in systemic capillaries is reflected in the peripheral venous channel is more or less of a similar magnitude as that in the pulmonary capillaries as reflected in the pulmonary arterial wedge or left atrial pressure tracings. Thus similar quantitative rise in the pressure in the systemic and pulmonary capillaries therefore had a different effect in as much as the systemic capillary pressure remained below the plasma osmotic pressure level. This explains the predominance of systemic congestion and oedema in cases of the constrictive pericarditis

Lack of pulmonary congestion and oedema —

The capacities of both the ventricles are more or less equally affected In mitral disease the left ventricle does not receive as much blood as the right ventricle ejects and therefore some blood is trapped in the pulmonary circulation But, in constrictive pericarditis the left ventricle receives and ejects as much blood as the right ventricle receives and ejects Therefore there is no hetero dynamism between the two ventricles as in the mitral disease This fact is reflected in low pulmonary artery pulse pressure This explains the lack of pulmonary congestion and oedema

However occasionally cases of constrictive pericarditis do get some symptoms of pulmonary congestion That may be due to some associated disease or in a very late stage of constrictive pericarditis or it may even be due to *inequality of pericardial restriction on each ventricle*

Fatigue exhaustion etc —

This is due to marked reduction or cardiac index which is due to the limited ventricular filling

Conclusion —

It is well known that the distensibility of the normal right myocardium is more than that of the left side By reasons of volume elasticity addition of the same volume of blood therefore will elevate the pressure less in the right side and more in the left side Identical rise of pressure how ever *on both sides* during the ventricular diastole indicates that the elasticity of the muscular element of both sides of the heart is greatly replaced by the rigidity of the fibrous element in the pericardium

INTRINSIC MYOCARDIAL DISEASE

Successful pericardiectomy in cases of constrictive pericarditis does *not always revert the deranged haemodynamics and pressure patterns* completely to normal This leaves room for the concept that the associated myocardial involvement plays a significant role in the production of the *characteristic haemodynamic picture Besides similar pressure tracings* have been obtained in intrinsic myocardial diseases and profound right sided failure

In intrinsic myocardial disease [18] the distensibility of the ventricle during diastole is limited and the ventricular late diastolic accomodation is inadequate In pronounced right sided failure hypervolemia associated with increased residual volume in the right ventricular cavities is mainly responsible to limit the right ventricular late diastolic accommodation

It is strongly felt that the intrinsic myocardial disease particularly myocardial fibrosis can produce similar pressure patterns In our experi

ence the patterns obtained in cases of myocardial fibrosis presumably of unknown etiology have been quite similar to those of constrictive pericarditis and none of the criteria from pressure pattern as proposed by several authors[14,16] before can be rigidly applied to distinguish one from the other

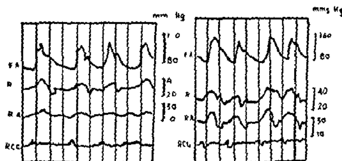


Figure 91

Pressure tracings in a case of myocardial fibrosis proved at autopsy. Insufficiency and failure showing large heart size causing tricuspid insufficiency (T I). The diagram shows rise in right atrial pressure with ventricular systole due to tricuspid insufficiency in a patient with hypertensive and arteriosclerotic heart disease (H A H D) and severe congestive failure. The pressures in the right ventricle and pulmonary artery are greatly increased

F A	Femoral Artery	R A	Right Atrium
R V	Right Ventricle	R C C	Electro cardiogram

Similar pressure patterns as described in constrictive pericarditis can be seen. However these cases of intrinsic myocardial disease are generally having auricular fibrillation and therefore typical M or W shaped right auricular tracing is conspicuously absent

There has been growing recognition of the similarity of haemodynamic pattern and clinical picture of constrictive pericarditis and the intrinsic myocardial disease. The mechanism of the production of similar pattern is the same as that in constrictive pericarditis except that in constrictive pericarditis the elasticity of the muscular element of ventricular myocardium is greatly replaced by the rigidity of the fibrous element in the pericardium while in myocardial fibrosis the elasticity is replaced by the rigidity of fibrous element in the myocardium itself. It is therefore implied here that all the characteristic haemodynamic patterns described in constrictive pericarditis is a function of the severity of the restriction in the ventricular distensibility irrespective of the cause thereof

As can be seen from the figure 92 it is difficult to distinguish intrinsic myocardial disease from chronic constrictive pericarditis by routine cardiac catheterization because the changes in pressures in the venous



Figure 92

Figure showing the mean pressures at different places in the right side of the circulation in cases of intrinsic myocardial disease as compared to normal

V I	Venous Pressure	I M D	Intrinsic Myocardial Disease
S V C	Superior Vena Cava	R V	Right Ventricle
I V C	Inferior Vena Cava	P A	Pulmonary Artery
R A	Right Atrium	P W	Pulmonary Wedge

side of the circulation are similar The ventricular pressure pattern the cardiac output and pulmonary vascular resistances are similarly affected in both the conditions

However certain findings in haemodynamic data and clinical picture are peculiar to the intrinsic myocardial disease itself In cases of myo

cardial fibrosis the tracings in the superior vena cava right auricle and the inferior vena cava were observed to have a general ventricular contour This is believed to be the result of the fact that the tricuspid valve has lost much of its function as a valve This is virtually marked tricuspid insufficiency The ordinary dynamics of the right auricle is partially masked This makes it possible to look upon the chambers as a continuous pipe like channel from the right ventricle to the right auricle and even extending to the superior vena cava and inferior vena cava The dilatation of the right ventricle due to the myocardial disease is thus considered primarily responsible for the observed marked tricuspid insufficiency

The auricular fibrillation and the profound enlargement of the cardiac chambers fluoroscopically can be considered consistent with the intrinsic myocardial disease

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CHAPTER II

VALVULAR LESIONS OTHER THAN MITRAL

LESIONS OF PULMONIC VALVE

PULMONIC STENOSIS

Although a vast majority of the cases of pulmonic stenosis are on the congenital basis, rheumatic infection attacks acutely the pulmonic valvular cusps but never leaves any deformity. True pulmonic stenosis can be due to inflammation and adhesion of the cusps.

Functional pulmonic stenosis have been rarely seen due to lodging of a large embolus extending from the outflow tract of the right ventricle to the pulmonary artery.

In the present series we did not have any opportunity of studying any case of acquired pulmonic stenosis, but it is quite reasonable to suppose that the pressure tracings in the right ventricle and the pulmonary artery would exhibit similar changes as that in congenital pulmonic stenosis.

A case reported by McGuire *et al*[34] of rheumatic pulmonic stenosis was associated with the stenosis of three other valves.

Biorck *et al*[1] in 1952 and Bean *et al*[13] in 1955 reported a very interesting combination of pulmonic valve stenosis, tricuspid valve lesion and a carcinoid of a small intestine with secondaries. It is believed that this carcinoid (argentaffinoma) and its metastases secrete large quantities of serotonin which is responsible for the pulmonary and tricuspid valve lesion as well as other signs of the syndrome.

Resistence of the pulmonic valve can be calculated as follows:—

$$\text{Resis. of Pulm. Valve} = \frac{(\text{RVsm}-\text{PAsm})}{\text{C.O.}} \times 60 \times 1333.4$$

RVsm = Right ventricular systolic mean pressure

PAsm = Pulmonary artery systolic mean pressure

Both of these can be calculated from the respective tracing.

C.O. = Cardiac Output

PULMONARY REGURGITATION

This is almost always functional in character and due to greatly increased pulmonary arterial blood pressure or pulmonary artery dilatation.

most commonly resulting from mitral stenosis on rheumatic basis and rarely of unknown etiology.

Functional pulmonary insufficiency can also result from the pressure of aortic aneurysm on the pulmonary artery or due to syphilis of the pulmonary artery.

Before the days of antibiotics cases of pulmonary insufficiency were reported due to bacterial endocarditis. No case of pulmonary rheumatic valvular deformity enough to interfere with its function has been reported in the last 10 years.

LESIONS OF AORTIC VALVE

AORTIC STENOSIS

The pressure tracing in a peripheral artery in severe aortic stenosis is very characteristic. However it has been shown in models that aortic opening has got to be reduced to ¼th its normal size before one begins to get a clinical picture of aortic stenosis. When the aortic orifice is reduced to about 60% of its normal size the cardiac output begins to be reduced. When the orifice reduces to about 20% of the normal the characteristic murmur becomes audible. When the aortic valve area goes down to 0.5 cm^2 it is considered a critical level producing markedly disturbed physiology.

Peripheral arterial tracing —

Peripheral arterial pulse tracing is of small amplitude—rises slowly and falls slowly. Prolonged systolic ejection period gives the pulse a form of a plateau. The pulse from its onset to the end of the plateau occupies about 0.24 seconds as compared to normal of 0.16 seconds. With more severe stenosis ascending limb has slight anacrotic notch. The position of this anacrotic notch on the ascending limb can indicate the degree of aortic stenosis. Greater the stenosis lower is the position of the anacrotic notch on the ascending limb. Occasionally the notch is very sharp which forms a double peak. The exact mechanism of the production of this notch is not known. However it is likely to be a sign of peripheral arterial wall reflection of the sudden recoil in the aortic wall when the aortic cusps offer marked resistance to flow after the initial period of systolic ejection[7].

The systolic pressure is diminished while the diastolic is generally normal resulting in a small pulse pressure.

The exact relationship of the central arterial pulse with the peripheral arterial pulse has not yet been understood[8].



Figure 93

Pressure tracing in the femoral artery alongwith the E C G showing the anacrotic notch on the ascending limb of the arterial tracing in a case of aortic stenosis

Central aortic pressure tracing —

The central aortic pulse recorded through the arterial catheter has a rounded or plateau shaped top with an anacrotic notch As the pulse is transmitted towards the peripheral arteries the systolic pressure increases normally and the peak becomes sharper In aortic stenosis central aortic pressure curves show more or less similar characteristics as that in the peripheral arterial tracing It shows initial steepness ending up in an anacrotic notch followed by slower rise reaching the peak relatively late in systole resulting into prolonged ejection phase The systolic pressure is generally less than normal The more severe is the stenosis the earlier is the anacrotic notch on the ascending limb The anacrotic notch of the central arterial tracing tends to be dampened as the wave is transmitted to the peripheral arteries The dicrotic notch is absent on the descending limb Thus three main changes occur with the development of the aortic stenosis —

i The normal amplification of the peripheral systolic pressure disappears in aortic stenosis
ii The systolic ejection period increases
iii The anacrotic notch appears even on the peripheral arterial pulse

Left ventricular pressure tracing —

The left ventricular pressure curve shows initial steep rise during the isometric contraction period and the peak is reached relatively early in systole The left ventricular systolic as well as diastolic pressure are elevated

The pressure gradient across the aortic valve —

Recent advances in left heart catheterization have made it possible to record simultaneously the left ventricular pressure tracing and a central

or peripheral arterial tracing Brock et al[34] have described the trans thoracic approach by inserting a small polyethelene catheter through a needle surgically introduced from behind into the left auricle By manipulating the catheter beyond the mitral valve left ventricular pressure tracings can be obtained Another method described to obtain the left ventricular pressure tracings is to pass a needle directly into the chamber from the area of the apex beat The third method which is under consideration at the present stage consists of threading a small polythelene catheter through a bronchoscopic needle inserted into left auricle trans bronchially and manipulating the tip of the catheter beyond the mitral valve All these methods must be undertaken only by a skilled surgeon who should be ready to perform thoracotomy immediately in case of complications The gradient may vary widely from 5 mm to 50 mm Hg depending upon the severity of aortic obstruction and efficiency of the ventricular myocardium

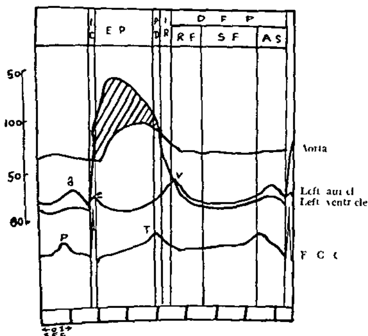


Figure 94

Simultaneous left auricular left ventricular and aortic pressure tracing (drawn theoretically) The shaded area indicates the marked ventriculoaortic pressure gradient during ventricular systole across the stenosed aortic valve The left auricular tracing excludes any significant degree of mitral disease

I C	Isometric Contraction	D F P	Diastolic Filling Period
E P	Ejection period	R F	Rapid Filling
P D	Proto-diastole	S F	Slow Filling
I R	Isometric Relaxation	A S	Atrial Systole

E C G Electro-cardiogram

Left atrial pressure tracing —

Left atrial mean pressure is elevated with a prominent a wave. This may be either a reflection of elevated left ventricular diastolic pressure due to aortic stenosis or it may be due to associated mitral stenosis. A method to exclude any coincident mitral stenosis would be Ry/V ratio lower than 1.6.[13] However it is noteworthy that Ry/V ratio in cases of pure aortic lesions with left ventricular failure can place these cases in mitral stenosis group.

Cardiac Output —

The reduction in cardiac output is relatively small until the stenosis becomes very severe.[11] With knowledge of cardiac output and trans valvular pressure gradient the aortic valve stenotic area can be calculated by Gorlin's hydraulic formula.[1]

Left heart catheterization —

Simultaneous tracing in the left ventricle by left heart catheterization and of central aorta by arterial catheterization is of immense value as a pre operative procedure in all cases of aortic stenosis. (Refer to figure 94) If the ventricular aortic pressure gradient, systolic ejection period and systemic output are known aortic valve area can be calculated.

AORTIC REGURGITATION (or INSUFFICIENCY)

The diagnosis of aortic regurgitation has hardly been a problem unless it is associated with several other valvular lesions. The difficulties and complications involved in the left sided catheter approach has limited the practice of obtaining the pressure pattern in the aorta or the left ventricle. In the present series we did not have the opportunity to study the central aortic or the left ventricular pressure tracing. However we have had several occassions to study the pressure pattern in the peripheral arteries.

Pressure pattern in brachial artery —

The ascending limb is sharply elevated from low diastolic level to somewhat elevated systolic level. The descending limb shows a markedly sharp fall of pressure up to the dicrotic notch beyond which the display of fall of pressure is more gradual. Sharply rising ascending limb indicates sharp rise of arterial pressure or rapid filling of the artery. The sudden fall of pre-dicrotic limb indicates sharp fall of pressure at the end of systole. This sudden rise and fall of pressure during systole gives the character of popularly called water hammer or collapsing pulse. The low diastolic pressure is partly because of backflow of blood towards the heart during diastole and partly because of the peripheral vaso dilatation.

Pressure pattern in femoral artery —

The general contour of the tracing is the same as that in the brachial artery but the femoral systolic pressure is higher than brachial by even 100 mm Hg instead of normal by about 20 mm Hg This is because of the fact that the femoral is in direct line with the aortic stream while the brachial issues from the aorta at an angle resulting into direct transmission of pressure head and also velocity head of aortic stream into the femoral artery

Central aortic tracing —

This shows rapid rise to a sharp elevated systolic peak followed by a sharp fall with poorly marked dicrotic notch

Left ventricular pressure tracing —

The systolic pressure is elevated as in the peripheral arteries The systolic peak is attained earlier in systole resulting into shortened isometric contraction period and the ejection phase The pressure then falls away steeply in late systole The maximum pumping therefore is accomplished in early systole Left ventricular diastolic pressure remains normal until late diastole when the pressure rises slightly because the regurgitating blood adds to the regular incoming blood from the left atrium into the relaxing left ventricle

Left atrial tracing —

The left atrial pressure tracing is not directly affected by aortic incompetence However when the left ventricular diastolic pressure rises the left atrial diastolic pressure also is elevated to a similar extent

Left heart catheterization —

Simultaneous recording of pressures in left ventricle left auricle and aorta can demonstrate aortic regurgitation

Absence of significant atrio ventricular pressure gradient and size and shape of v wave can exclude the mitral disease Elevated left ventricular pressure during ventricular diastole over that in the left auricle presumably indicates that the blood is regurgitating from the incompetent aortic orifice

Cardiac Output —

The cardiac output is maintained normal as long as the left ventricle is competent Ultimately the left ventricle begins to fail sometimes suddenly and the cardiac output falls below normal

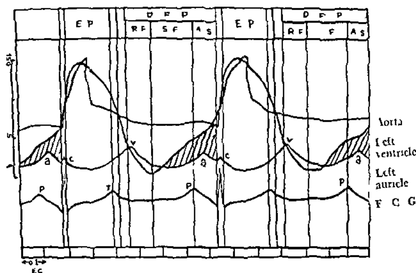


Figure 95

Simultaneous left auricular, left ventricular and the aortic pressure tracings (drawn theoretically). The shaded area indicates that the left ventricular pressure actually exceeds the left auricular pressure during late diastole presumably due to the large volume of blood regurgitating through the incompetent aortic valve. The aortic tracing shows prompt rise and fall of pressure indicating collapsing quality. The left auricular tracing excludes any significant degree of mitral disease.

E P	Ejection Period	S F	Slow Filling
D F P	Diastolic Filling Period	A S	Atrial systole
R F	Rapid Filling	E C G	Electro cardiogram

LESIONS OF THE TRICUSPID VALVE

TRICUSPID INSUFFICIENCY

Tricuspid insufficiency of functional origin is much more common than that of organic origin.

Conditions associated with the right ventricular hypertrophy and dilatation of the tricuspid ring give rise to the tricuspid insufficiency with the advance of the disease. Peculiar to the tricuspid leaflets which are too small to close the orifice completely, slight tricuspid insufficiency is physiologically present even in normal beings. In the absence of effective right ventricular contraction, closure of the tricuspid orifice is not complete. Atrial fibrillation can also contribute to insufficient closure of the tricuspid orifice. Besides, enlargement of the right ventricle retracts the papillary muscles and chordae tendinae from the tricuspid leaflets. Associated dilatation of the right ventricle enlarges the tricuspid ring and orifice.

Organic tricuspid insufficiency is occassionally noted in cases of rheumatic mitral or aortic valvular disease. Rarer causes of tricuspid insufficiency described in the literature are traumatic, bacterial endocarditis and even congenital.

Right atrial superior vena caval inferior vena caval and jugular venous tracing —

In cases of the right ventricular congestive failure with normal tricuspid valve there is a fall of pressure in the right auricle during early ventricular systole due to descent of the tricuspid valve but with insufficient tricuspid valve during ventricular systole blood regurgitates in the right auricle from the right ventricle leading to the increased diastolic volume of blood in the right auricle As a result of this persistent right auricular stasis and the impact of regurgitant stream the pressure in the right auricle and the neighbouring venous system increases markedly giving rise to well known v wave of tricuspid insufficiency This is sometimes called ventricular venous pulse or ventricularization of the venous pulse For the same reasons the slope of the descending limb of v wave becomes rapid giving rise to a plateau or even a positive wave between v and c waves of the subsequent beat [4] This explains the mechanism of the clinically observed systolic expansion of the external jugular veins or internal jugular veins near the ear lobe

The striking feature however is the reversal of central venous pressure gradient during the ventricular systole The normal flow is restricted during diastole Moreover it may be possible in the course of cardiac catheterization to demonstrate higher v wave in the right auricle than that in the subclavian vein

The figure 96 shows that in tricuspid insufficiency there is a reversal of a pressure gradient between the superior vena cava and the right auricle during the entire ventricular systole [16] The right auricular pressure is elevated often to a mean pressure of 10 to 25 mm Hg and increases much higher during exercise The reversal of flow from the right auricle to superior vena cava during ventricular systole increases during exercise

The height of v wave will also be controlled by the dilatation of the right atrium by the volume elasticity properties of its musculature With greatly dilated right auricle the systolic regurgitant stream has little effect on its large content

The presence of the atrial fibrillation will also modify the right atrial tracing Auricular fibrillation increases the right atrial and venous stasis and will also prevent the effective closure of the tricuspid valve as atrial systole is necessary for the complete closure of the tricuspid valve Therefore in the presence of auricular fibrillation and right heart failure v wave may be accentuated despite normal tricuspid valve However v wave will be much more elevated with the tricuspid insufficiency

Right ventricular pressure tracing

The right ventricular systolic diastolic and mean pressures are elevat

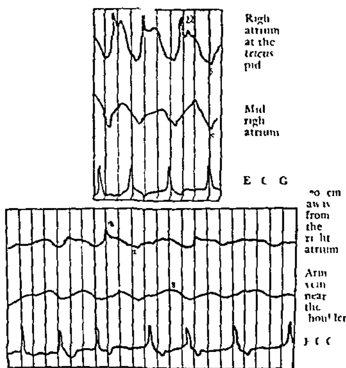


Figure 96

Figure showing the reversal of central venous pressure gradient during the ventricular systole in tricuspid insufficiency The systolic venous pressure increases from 18 to 24 mm Hg as one goes from the arm veins to the right atrium near the tricuspid. E C G Electro cardiogram

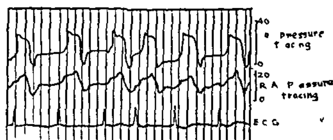


Figure 97

Right ventricular (R V) pressure tracing showing the typical pattern with an elevated end diastolic pressure and the right atrial (R A) pressure tracing showing the tricuspid insufficiency in a case of intrinsic myocardial disease with slight congestive heart failure

E C G Electro cardiogram

ed The right ventricular pressure pattern in general is similar to that found in chronic constrictive pericarditis The early diastolic dip in the right ventricular tracing slightly precedes the fall of large v wave in the

right auricular tracing simultaneously recorded because in early diastole the atrio-ventricular pressure gradient is the highest and the right ventricle is able to receive the maximum amount of blood An elevation of the right ventricular diastolic pressure if present denote the right ventricular failure

The pulmonary flow initially is diminished by the amount of blood that regurgitates in the right auricle during systole The larger volume of blood in the right auricle subjects the right ventricle to a higher filling pressure and the right ventricle is dilated further to accommodate extra blood The greater the right ventricular dilatation greater the degree of tricuspid incompetence and therefore greater the leak Ultimately the situation gets stabilized

Associated lesions

The dynamics of mitral and/or aortic valvular lesions may be modified by the presence of functional tricuspid insufficiency In cases of mitral stenosis the pulmonary resistance were found to be higher when accompanied by functional tricuspid insufficiency

Dye injection

In tricuspid insufficiency part of the Evans blue dye when injected in the right ventricle through the distal lumen of a double lumen catheter with the proximal lumen in the right auricle can be recovered from the proximal lumen Similar technique has been attempted in the experimental mitral insufficiency

TRICUSPID STENOSIS

Organic tricuspid stenosis is invariably due to rheumatic fever This in almost 90% of cases is associated with other valvular lesions mitral or aortic Isolated tricuspid stenosis reported in the literature have been very rare [10] Occassionally tricuspid stenosis is associated with tricuspid insufficiency The other causes of tricuspid stenosis are bacterial vegetations thrombi or tumors projecting in to the tricuspid orifice and even on congenital basis The most reliable method of diagnosing or excluding tricuspid stenosis is cardiac catheterization

Right atrial superior vena caval inferior vena caval and jugular venous tracing —

Contrary to tricuspid insufficiency increased diastolic filling of the right auricle in tricuspid stenosis is due to incomplete emptying of the right auricle during ventricular diastole

The large volume of atrial blood in the late ventricular diastole induces more forceful atrial contraction giving rise to marked a wave

of atrial systole The descending limb of a wave shows more gradual fall because of blood flowing through the narrowed tricuspid orifice as compared to rapid or sudden fall in tricuspid insufficiency

Besides obstruction at the tricuspid orifice can cause the back flow of blood into the neighbouring veins during atrial systole This explains the mechanism of clinically observed cervical venous pulsation during atrial systole This is sometimes called atrial venous (or atrialization of venous) pulse This is also occassionally observed in inter atrial septal defect The c wave is generally not remarkable

The mean right auricular pressure is also elevated markedly in tricuspid insufficiency [16]

The gradual fall of the descending limb of v wave in the right auricle is the most important single factor in the diagnosis of the tricuspid stenosis [1] Adequate number of cases of tricuspid stenosis have not been catheterized to work out Ry/V' ratio With the surgical relief of tricuspid stenosis the transtricuspid diastolic pressure gradient and the slope of descending limb of the right auricular v wave can be brought closer to normal

However the most marked change and the diagnostic feature of tricuspid stenosis is the increased pressure gradient between the right auricle and the right ventricle during ventricular diastole [1] If simultaneous pressure tracings are taken in the right auricle and the right ventricle with a double lumen catheter it can be demonstrated that the pressure in the right auricle is considerably higher than the right ventricular diastolic pressure during the entire diastole This is conspicuously absent in tricuspid insufficiency

Right ventricular pressure tracing

The systolic and the diastolic pressure should be normal but practically they are higher because of associated mitral disease

Combined tricuspid lesions

Expert clinicians do observe the double cervical venous pulsations—one systolic (during ventricular systole) and the other pre systolic (during atrial systole) The right atrial tracings of these cases will demonstrate giant a wave of tricuspid stenosis and big v wave of tricuspid insufficiency

Tricuspid valve stenotic area

Knowing the pressure gradient across the tricuspid cardiac output

and the right ventricular diastolic filling period tricuspid valve stenotic area can be calculated by Gorlin's hydraulic formula.

Cardiac output

It is hardly necessary to mention the diminution in cardiac output. Resistance of the tricuspid valve can be calculated as follows:

$$\text{Resistance of the tricuspid valve} = \frac{(RAm - RVdm)}{CO} \times 60 \times 1332 \cdot 4$$

where RAm is right auricular mean pressure
RVdm is Right ventricular diastolic mean pressure
and CO is Cardiac output.

As the right auricular and right ventricular pressure tracings are easily available the value obtained is free of such assumptions.

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CHAPTER III

HYPERDYNAMIC CIRCULATORY STATES

HIGH OUTPUT FAILURE

[The discussion of high output failure has been included here because now a days the most popular and correct available method of calculating the cardiac output is by right heart catheterization]

Physiological increase in cardiac output can be caused by several factors like muscular exercise digestion of food excitement therapeutic infusions pregnancy high altitudes etc etc However discussion here is limited only to the pathological conditions associated with high output failure

Most important conditions where the diagnosis is likely to be missed are the following

i Hyperthyroidism
ii Acquired left to right shunts (Areterio venous fistulla)
iii Anaemia and Beriberi
iv Severe pulmonary emphysema
v Pagets disease of bones certain liver diseases etc

When any of these conditions is accompanying any mitral disease the haemodynamic findings are so much modified that the correct evalua tion of the patient is not possible

Hyperthyroidism

It is of paramount importance to exclude hyperthyroidism in a case of mitral stenosis before evaluating for surgery It has happened in the past that in cases of mitral stenosis accompanying hyperthyroidism has been diagnosed during cardiac catheterization

Pure hyperthyroidism markedly increases the cardiac output in order to meet with the increased metabolic needs of the body and heat produc tion At the same time the elevated cardiac output keeps the heart under great strain Haemodynamics in a case of heart failure will be completely modified by the complicating hyperthyroidism Thus cardiac output in a hyperthyroid individual with the profound congestive heart failure can be high in absolute values

Acquired left to right shunts

(Arterio venous fistulla)

One case of acquired left to right shunt at the aorto pulmonary level probably due to syphilitic aortitis was studied in the present series. The cardiac catheterization findings were more like those of patent ductus arteriosus except that the point of arterialization of blood was somewhat at a lower level i.e. at the root of the pulmonary artery. Besides the calculated cardiac output was close to 9 liters. The closure of the fistula could not be attempted because the patient died perhaps due to the complication of hypothermia at the operation table. The elevated cardiac output in these cases is due to increased stroke output and tachycardia[9,13]. The mechanism of causation of high cardiac output consists of augmented venous return and lower systemic resistance. When congestive failure sets in cardiac output somewhat falls but still it is higher in absolute values.

The diminished systemic resistence is because the arterial blood finds it easier to flow via the fistula through the venous channels with lower resistance rather than passing through the normal arterial channels. Subsequently blood volume increases raising the venous pressure still further resulting into augmented cardiac output. Eventually a state of balance is reached.

Anaemia and beriberi

These two coditions can be discussed together because the underlying mechanisms of the elevated cardiac output appears to be very similar in each of them. Reduction in peripheral resistance because of the vaso dilatation of the systemic capillaries due to accumulation of acid metabolites is a common factor in both of these conditions.

In beriberi these acid metabolites are pyruvic and lactic acid because of lack of thiamine which is necessary for carboxylase the enzyme essential to oxidise these acid metabolites. In severe anaemias it is tissue anoxia which lead to the accumulation of these metabolites[1]. In either case these metabolites act directly locally or reflexly on peripheral capillary dilatation. These peripheral capillary dilatation has the haemodynamic effect simulating multiple left to right small shunts. The marked diminution of the obstructive element at the systemic capillaries results into acceleration of the speed of circulating blood and therefore circulation time diminishes markedly. Venous return increases at the same time resulting into elevated cardiac output.

Severe pulmonary emphysema

If the pulmonary function with all its reserve is not enough to maintain the normal oxygen supply to the tissues the resulting tissue hypoxemia reduces the peripheral resistance thereby increasing the speed of circulating blood and venous return[2]. This results into elevated cardiac output.

However along with this hypoxemia if the pulmonary resistance increases greatly the speed of circulating blood may not increase at all The pulmonary capillaries then behave as obstruction and prevent the speeding of circulation In practice cardiac output is frequently diminished or normal in cases of cor pulmonale

Paget s disease

The resistance to the blood flow through the actively diseased bones is diminished resulting into hyperkinetic circulatory state This increases venous return The bony channels act as multiple arterio venous fistulae

Liver diseases

Normally the liver detoxicates vaso depressor substances In extensive liver destruction these toxic substances accumulate in the body giving rise to vaso dilatation The mechanism is more or less the same as in beriberi

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APPENDIX

Number	Name	Age	Sex	Surface Area	History and duration of Rheumatic Heart Diseases in years	Dyspnoea: Maximum In The Past	Dyspnoea: At Admission In Hospital	Orthopnoea	Paroxysmal Nocturnal Dyspnoea	Fatigue	Haemoptysis	Oedema at Admission	Duration Of Symptoms in years	Hepatomegaly	Emboli	Functional Group (N Y Heart Association)	Auscultation: Mitral Systolic murmur	Auscultation: Mitral Diastolic murmur	Auscultation: Aortic	Auscultation: Pulmonary	Auscultation: Tricuspid	E C G: Rhythm	E C G: Left Auricle	E C G: Ventricle Right Or Left
101	I M	[illegible]	F	1 1		+	+	+	1+	0	1+	0		0	0	III	1+	+	0	1 +	0	R S R	+	R V +
10[illegible]	R E	3[illegible]	F	1 5	10	+	1+	0	0	1+	0	0	10	+	0	II	1+	+	±	1 +		R S R	1+	R V +
103	R I	[illegible]	F	1 43	8	+	1+	0	0	0	±	±	16	0	0	II		+	1+	0	0	R S R A F	+	R V +
104	G K	[illegible]3	M	1 6	19	+	1+	1+	1+	0	+			3+	0	III	+	3+		1 +	S+	R S R	+	R V +
10[illegible]	B [illegible]	–	F	1 43	6	3+	+	+	1+	±	±	±	20	+	1+	III	1+	+	[illegible]	1 1+	S+	A F	+	R V +
106	S L	[illegible]	F	1 40	8	4+	+	+	0	1+	+	1+	8	1+	1	III	0	+	[illegible]	1 1+	0	R S R	+	R V +
10[illegible]	K I	–	F	1		3+	1+	0	0	[illegible]	0	0		+	0	III	1+	+	[illegible]	1 1+	S+	R S R	+	R V +
[illegible]	[illegible] C	4	M	1 64	10	+	1+	1+	0	[illegible]	+	1+		0	0	II	+	+	[illegible]	1 +	0	R S R	1+	R V 1+
[illegible]	[illegible]	–	M	1 6	6	5+	1+	+	0	0	0	+	6	+	0	II	0	+			0	R S R	1+	R V 1+
[illegible]	G I	[illegible]	M	1 8	4	1+	1+	0	0	1+	+	0	4	[illegible]	[illegible]	II	0	+	0	1 1+	[illegible]	R S R	1+	R V 1+
111	[illegible] I	[illegible]	F	1 4	11	4+	+	1+	±	0	+	1+	4	+	1	II	0	1+		P2 +		R S R Old [illegible]	+	R V 1+
11[illegible]	G D	[illegible]	F	1 [illegible]	6	3+	2+	±	1+	0	1+	1+		1+	0	II	1+	3+	0	P2 1+	[illegible]	R S R	+	R V +
113	S B	51	F	1	1	3+	+	1+	[illegible]	+	0	3+	11	3+	0	III	1+	+		1 1+	S+	A F	+	R V +
11[illegible]	F M	[illegible]	F	1 [illegible]	4	4+	4+	4+	2+	1+		1+	4	[illegible]	±	II	1+	+		1 +	S+	R S R	+	R V +
116	H H	3[illegible]	F	–	30	+	1+	0	[illegible]	0	0	[illegible]	16	0	0	II	0	+	0	0	0	A F	1+	0
11[illegible]	I M	[illegible]0	F	–	[illegible]	+	1+	1+	[illegible]	0	1+	±	[illegible]0	0	0	II	0	+		1 1+	0	R S R	+	R V +
118	W A	41	F	1	31	3+	1+	1+	1+	±	0	0	4	0	0	II	0	+	[illegible]	1 1+	[illegible]	R S R	+	R V 1+

Appendix I Group I (101 to 118)

History and clinical data in cases having pure mitral stenosis The group was studied by cardiac catheterisation and also pressures were recorded in pulmonary artery and left auricle before and after mitral commissurotomy by direct needle puncture at the operation

The results are expressed from 1+ to 4+

- P2 Pulmonary second sound
- A F Auricular Fibrillation
- D Diastolic
- R S R Regular Sinus Rhythm
- R V Right Ventricle
- S Systolic

	Fluo oscopy							Duration Of M d cal Th rapy				P ripheral Ar ry Pressure in mm of Hg						Pulmonary Artery pressure in mm of Hg (By Catheter)						Left Auricular Mean Pressure (Direct Punc ture) in mm of Hg		Mitral Insufficiency As Judged from LA Curve	
Number	Left Auricle	L ft V ntricle	Right Ventricle	Right Auricle	Pulmonary Artery	Mitral Valve Calcification	Aortic Valve Calcification	Less Than 1 Year	More Than 1 Year	Circul tion Time in seconds	Venous Pressure in mm of Water	Systolic	Diastolic	M n	Blood Pressure (By Brachial Cuff)	Right Auricular Mean Pre u e mm of Hg	Right Ventricular Mean P e ure in mm of Hg	Systolic	Diastolic	Mean	Pulmonary Artery Wedge Mean Pressure in mm of Hg	PAm PWm Gradient	Mitral Insufficiency As Judged From PW Curve	Pre op	Post op	Pre op	Post op
101	+	0	1+	0	1+	0	0	+	–	1	1L	98	5		11 /0	–		6	3	39	–	–	0	–	–	0	0
10'	+		+	±	+	1+	0	+	–	3	90	–	–	–	101 66	10	)		40	59	30	0	0	28	13	0	0
10	+	0	1+	0	1+	0	0	–	+	1	4	1	7	9)	11_/70	10	)	40	1	–	1	6	0	4	0	0	±
101	+	1+	+	1+	+	1+	0	+	–		100	11	8	9)	11 85	6	4	85	0	6	3	3)	0	3	5	0	0
105	+	0	+	1+	1+	0	0	–	+		3	98	t	3	110 60	4	)	3.	1	25	–	–	–	28		0	0
106	+	±	+	+	+	1+	0	–	+	[illegible]	95	9	0	8	1 80		4	100	60	65	0	4	0	–	–	–	–
10	+	±	+	0	+	0	0	+	–	14	91	38	6	91	100 8	11	5	58	34	48	30	18	0	50	33	1+	+
108	+	0	1+	0	1+	0	0	–	+	18	9	1	4	81	–		1	31	11	–	1	5	0	27	1	0	0
109	1+	0	0	0	1+	0	0	–	+	14	110	–	–	–	–		0	47	22	25	0	5	0	–	–	–	–
110	+	0	±	0	1+	0	0	+	–	17	118	11)	76	8)	–	4	1	[illegible]		16	1	4	0	–	26	0	1+
111	+	0	1+	0	1+	0	0	–	+	11	60	11	63	–	–	10	14	56	10	26	1	14	0	30	–	1+	0
11	+	0	+	0	+	0	0	–	+	1	9	11	6	8	9 70	6	28	7	38	47	34	13	0	41	33	0	1+
113	+	)	+	0	+	0	0	–	+	16	11	130	9	1 0	–	4	4	30	14	25	1	4	0	78	1	0	0
11	4+	1+	+	1+	+	1+	0	–	+	1)	81	–	–	–	1 /	1	31	53	1	0	–	–	–	40	[illegible]	0	0
116	1+	(	0	0	0	0	0	+	–	[illegible]	3	–	–	–	93/ 0	–	[illegible]	–	–	–	–	–	–	1	1	0	0
117	2+	0	+	1+	+	0	0	–	+	13	85	–	–	–	11 8			6	25	1	–	–	–	3	[illegible]	0	r
118	+	0	1+	0	1+	+	0	+	–	19	4	1 0	6	0	108 0	4	10	9	14	–	1		0	4	4	0	0

Append x I Group I (101 to 118)

Investigational and haemodynam c data in cases hav ng pure m t al stenos s The g o p was stud ed by cardiac catheterization and also pressures were recorded n the pulmo ary artery and left aur cle befo e and after m tral comm ssurotomy by direct needle punctu e at the operation table

The results are expressed f om 1+ to 4+

Pre op Pre operat ve

Post op Post operative

Number	(By Direct Puncture) Pulmonary Artery Pressure in mm of Hg — Systolic: Pre Operative	Systolic: Post Operative	Diastolic: Pre Operative	Diastolic: Post Operative	Mean: Pre Operative	Mean: Post Operative	Arterial Oxygen Saturation	Air Ventilation Index	Oxygen Consumption Index	Arterio Venous Oxygen Difference	Cardiac Output in Litres	Cardiac Index	Stroke Volume in c c	Stroke Index	Total Pulmonary Resistance	Pulmonary Arteriolar Resistance	Mitral Resistance	Systemic Resistance	Right Ventricular Pressure Work	Left Ventricular Pressure Work	Mitral Valve Area (By Gorlin s Formula)	Mitral Valve Area As Judged By Surgeon: Pre Operative	Mitral Valve Area As Judged By Surgeon: Post Operative	Regurgitant Jet Felt By The Surgeon: Pre Operative	Regurgitant Jet Felt By The Surgeon: Post Operative	Remarks
101		6	0	[illegible]	[illegible]	4	00	3 0	1 4	34	3 0		1. 48	T	4 0	60X	0	16.0	1 7	16	0	F g r	Fl g	0	0	
10	6	4	8	15			31	9	8 9	1 31	08	1 1	6. 49	5	1.3	03	108	1010	6 97	—	0 3	Fl g	F g	0	0	
103	75	[illegible]	1	18	[illegible]	—	96	1 0	110	6 09	8	1 11	41	9	8.7		6	311	5 0 41	1 64	0	F n	Fl a		+	
104		8	7	0	67	4	94	81	144	6 4	3 51	1 93	[illegible]		0 179	696	606	1 1 0	4	4	8 f	F g	Fl g	1	0	
10	40	40	0	[illegible]	1	38	91	0	179	08	7		[illegible]	0	4		—	2. 1	1	38	—	F L	F g	0	0	
106	107	11		60	67	85	94	4	113	8 79			1 80	r	3 60	1435	116	708 1		1	0 49	Fing	Fl g r	0	F l t J l	
107	190	100	4		63	3	96	4 16	133	4 51	4 4		11 4		.0	8 0	340	50 1	0 1 6.	3 3	0 86	Fl ger	Fl gc	0	1+	
[illegible]		[illegible]	16	11	4	18	86	1	114	4 18	4 47		3		40	781	111	1 14.0	0 8	3 1	1	F ger	Fl g	0		
[illegible]		[illegible]	8	11	41	18	96	1 11	118	34	4 6		6 3		4	41	89	1 1 11 6	0	—	0 9	Fl g	Fl g r	0	0	
[illegible]	4	4		[illegible]	30	34	8	3 91	14	4 4	40	3 31	64	11	78	10	136	1140	0 88	4 0	1 16	F L	Fl g	0	0	
111	7	6.		0	47	41	—	3 61	110	5 33	50	4	4	3	608	11		33 1	~ 0	—	1 38	Fl g	Fl	0	)	
11	66	66	4	44	40	3	—	3 1	113		3 8		18 40		0	1	70	1 1794 1		—	0 4	Fl g	Fl g r	0		
11		4	47	29	9	38	00	0	—		1 3		4 41	9		0		3 1	1 1	—	71	F g	Fl g r	0	+	
115	7	4	[illegible]	39	0	3	—	—		—			—		—	—	—	—	—	~	—	Fl g r	Fl g r	0		
116	44	41	~	1	30	8		~	~		~	—	—	~	—	—	—	—	~	~	~	Fl ger	Fling r	0	0	
117	1	0	7	27	[illegible]	48	—		—				—	—	—	—	—	—	—	—	—	Fing	Fl g r	0	0	I [illegible] der I pad (bis)
118	40	37	40	41	78	73	9	3 8	116	4 4	3 9		63	—	—	4	14	70 14	0 60	87	90	Fl g r	F g	0	0	

Append x I Group I (101 to 118)

Haemodynam c and Surgical data in cases having pure m tral stenosis The group was stud ed by cardiac catheterization and pressures were obtained in pulmonary artery and left auricle before and after m tral commisurotomy by direct needle puncture at the operation table

Pre-op—Pre operative Post op—Post operative

Number	Name	Age	Sex	Surface Area	History and duration of Rheumatic Heart Diseases in years	Dyspnoea: Maximum In The Past	Dyspnoea: At Admission In Hospital	Orthopnoea	Paroxysmal Nocturnal Dyspnoea	Fatigue	Haemoptysis	Oedema at Admission	Duration Of Symptoms in years	Hepatomegaly	Emboli	Functional Group (N Y Heart Association)	Auscultation: Mitral Systolic murmur	Auscultation: Mitral Diastolic murmur	Auscultation: Aortic	Auscultation: Pulmonary	Auscultation: Tricuspid	E C G: Rhythm	E C G: Left Auricle	E C G: Ventricle Right Or Left
1 5	G M	41	F	1 69	21	+	1+	1+	1+	1+	1+	+	21	3+	0	III	1+	4+	0	)	0	Ar	+	Rv
1??	(D	41	M	1 6	1	3+	+	3+	1−	)	0	0	1	+	0	III	±	+	D+	0	0	[RSR	+	R
128	D J	51	F	1 4	0	+	1+	+	0	+	1+	3+		1+	0	III	1+	+	0	D+	0	Ar	+	Rv
1 9	C F	36	F	1 6	13	4+	+	+	1+	1+	+	4+	13	1+	+	III	+	+	D+	0	0	Ar	+	Rv
130	P P	5	M	1	1	+	1+	+	0+	0	+	0	1	(	0	II	0	+	0	P	0	Ar	+	Pv
131	T J	41	M	1 98	(	+	1+	+	+	1+	0	±		1+	0	III	±	+	S+	P	0	\	+	R
13	R I	48	M	1 8	−	+	+	1+		+	1+	+	9	1+	0	II	1+	3+	0	0	0	RSR	+	Rv
1 3	F E	48	F	1 5	⌐	+	1+	+	0	1+	0	+		+	+	III	+	3+	0	0	0	RSR	+	R
134	K J	28	M	−	−	1+	1	1+	0	)	3+	+	−	0	0	III	+	+	0	D+	0	Ar RSR	+	Rv
13	P W	41	M	1 9	1	+	1			I		0	(	1+	0	III	3+	+	0	P	)	Ar	+	Rv

Appendix I Group 2 (126 to 135)
History and clinical data in cases having predominant mitral stenosis and mild mitral insufficiency The group was studied by cardiac catheterization and also pressures were recorded in pulmonary artery and left auricle before and after mitral comissurotomy by direct puncture at the operation table

Number	Name	Age	Sex	Surface Area	History and duration of Rheumatic Heart Diseases in years	Dyspnoea: Maximum In The Past	Dyspnoea: At Admission In Hospital	Orthopnoea	Paroxysmal Nocturnal Dyspnoea	Fatigue	Haemoptysis	Oedema at Admission	Duration Of Symptoms in years	Hepatomegaly	Emboli	Functional Group (N Y Heart Association)	Auscultation: Mitral Systolic murmur	Auscultation: Mitral Diastolic murmur	Auscultation: Aortic	Auscultation: Pulmonary	Auscultation: Tricuspid	E C G: Rhythm	E C G: Left Auricle	E C G: Ventricle Right Or Left
1 1	FM	48	F	1 5	41	+	+	−	)	1+		1+	40	+	0	III	+	3+	)	0	0	AF	+	Rv
1	SR		F	1		+	1+	+	0	1+	0	)	1	1+	+	II	1+	+	0	0	0	Ar	+	Rv
13	CI	−	F	1 6		3+	+	+	1+	0	0	1+		+	1+	III	0	1+	(	0	0	A	+	0
14	CI	31	M	1 9?		1+	1+	0	0	+	+	0		3+	0	III	1+	+	0	0	0	\	+	Rv
15	HK	−	F	−	1	+	1+	0	0	0	0	)	21	3+	0	II	1+	+	1+	0	0	Ar	+	0

Appendix I Group 3 (151 to 155)
History and clinical data in cases having no mitral stenosis The group was studied by partial catheter a tion and was erroneously subjected to mitral commissurotomy The absence of mitral stenosis was judged at the time of surgery
S Systolic D Diastolic

Number	Left Auricle	Left Ventricle	Right Ventricle	Right Auricle	Pulmonary Artery	Mitral Valve Calcification	Aortic Valve Calcification	Less Than 1 Year	More Than 1 Year	Circulation Time in seconds	Venous Pressure in mm of Water	Systolic	Diastolic	Mean	Blood Pressure (By Brachial Cuff)	Right Auricular Mean Pressure mm of Hg	Right Ventricular Mean Pressure in mm of Hg	Systolic	Diastolic	Mean	Pulmonary Artery Wedge Pressure in mm of Hg	PAm PWm Gradient	Mitral Insufficiency As Judged From PW Curve	Pre op	Post op	Pre op	Post-op
	Fluoroscopy							Duration Of Medical Therapy				Peripheral Artery in mm of Hg (By Catheter)						Pulmonary Artery Pressure in mm of Hg (By Catheter)						Left Auricular Mean Pressure (Direct Puncture) in mm of Hg		Mitral Insufficiency As Judged From LA Curve	
1?6	4+	±	1+	1+	3+	+	0	—	+	—	10)	—	—	—	—	3	3	41	18	0	10	0	—	31	17	—	—
1?7	+	1+	+	0	+		)	+	—	19		)	)	0	—	4	[illegible]	6?	[illegible]	?0	[illegible]	1	—	11	11	—	+
1 8	3+	+	+	1+	J+	+	0	—	+	5	10	141	K	10	—	11	-	4	14	34		K	+	-		+	+
129	3+	±	2+	+	1+	+	0	—		—	—	1?4	64	8?	—	[illegible]	1	40	0	8	1	11	+	3?		+	+
30	1+	0	1+	0	±	0	t	—	+	13	1	10?	51		1 /?0			—	—	—	—	—	—	1		0	+
131	3+	1+	3+	1+	+	+	)	—	+	[illegible]	140	34	68		—	9	46	0	[illegible]	68	31	[illegible]	0	38	[illegible]	0	+
~		1+	1+	±		0	0	+	—	0	115	1?0	60		91?	3	0	60	[illegible]	)	)	1	0	[illegible]	71	0	0
		0	+	0	1+	+	0	—	—	[illegible]	130	1	[illegible]		—	[illegible]	8		0	41	17	1	—		[illegible]	0	0
1	+	0	+	0	1+	+	)	—	+		—			— 11	6		18	96		6	—		—	J		+	+
13.	4+	0	+	+	1+	+	0	—	+	) 1	)	1 1	0		—	—	0	(	1		18		+	[illegible]		+	+

[redacted] Appendix I Group 2. (126 to 135)

Investigational and haemodynamic data in cases having predominant mitral stenosis and mild mitral in sufficiency The group was studied by cardiac catheterization and also pressures were recorded in pul monary artery and left auricle before and after mitral commissurotomy by direct needle puncture at th operation table

151	3+	±	+	1+		1+	0	—	—	11	9			67	—	11	4			4	7	8		—	—	—	—
15	4+	1+	+	+	+				—		—	1			—	1	0	40		(	--	13	0	3	—	+	—
15?	3+	1+	1+	0							F	9			—		11	11	[illegible]	18	10	8		1	1	0	0
1?4	(	1+	1+	1+	1+	3+			+	1 1		1			1 1	,		4?		41			—	?		0	0
15?	3+	1+	+	1+	+	0			—	1		1 1		1(	110 ✓	6	19		0	11	—	—	—		3	+	+

[redacted] Appendix I Group 3 (151 to 155)

Investigational and haemodynamic data in cases having no mitral stenosis The group was studied by partial cardiac catheterization and was erroneously subjected to mitral commissurotomy The absence of mitral stenosis was judged at the time of surgery

Pre op Pre operative Post op Post operative

Case	Pressure in mm of Hg Pulmonary Artery (By Direct Puncture): Systolic, Pre Operative	Systolic, Post Operative	Diastolic, Pre Operative	Diastolic, Post Operative	Mean, Pre Operative	Mean, Post Operative	Arterial Oxygen Saturation	Air Ventilation Index	Oxygen Consumption Index	Arterio Venous Oxygen Difference	Cardiac Output in Litres	Cardiac Index	Stroke Volume in c c	Stroke Index	Total Pulmonary Resistance	Pulmonary Arteriolar Resistance	Mitral Resistance	Systemic Resistance	Right Ventricular Pressure Work	Left Ventricular Pressure Work	Mitral Valve Area (B) Gorlin's Formula	Mitral Valve Area As Judged By Surgeon: Pre Operative	Mitral Valve Area As Judged By Surgeon: Post Operative	Regurgitant Jet Felt By The Surgeon: Pre Operative	Regurgitant Jet Felt By The Surgeon: Post Operative	Remark
[illegible]	6	—		—	50	—	90	4 44	1 ^8	8	4 [illegible]		—	—	1	36^	148	—	1 1	—	—	F ¼	F ng	1+	1+	
7	93	9	50	50	67	65	91	43	1[illegible]	5 3	4 8	49	58	37	91	4 0	49[illegible]	1[illegible]	1 6[illegible]	—	—	F ¼ g	Fl g	1+	1+	
8	60	53	0	6	0	4	94	8	116	6 3	60	1 8	[illegible]	19	6 1	191	680	3150	0	—	—	F ¼ r	F g	1+	1+	
9		65	4	5	50	55	94	38	109	5 36	9	1 [illegible]	4	8	68	301	465	190	0 58	—	—	F 1¼ g	F ¼ ng	1+	+	
9	33	43	1	[illegible]	4	[illegible]8	—	4	14	4 65	5 4	3 05	43	[illegible]8	—	—	—	—	—	—	—	F 1	Fl g	1+	1+	
1	8	6	5	48	60	55	—	4 8	11	8 13	[illegible]	1 39	[illegible]9	1	[illegible]107	1 1	89	[illegible]10	1 6	—	—	F ¼ g	F n[illegible]	1+	1+	
[illegible]	95	110	0	0	6	7	100	—	—	5 8	4 3		4 0	[illegible]	5[illegible]		8 4[illegible]	1	0 98	—	—	F ¼ g	F 1½ g	2+	1+	
[illegible]	50	5		[illegible]	6	43	[illegible]	0	11	6 4	46	19	—	—	9	684	[illegible]1	1966	0 6	—	—	F ¾ g	F g	1+	¾+	
[illegible]	68	60	0	30	40	40	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	F ¼ g	F g	1+	1+	
[illegible]	85	3	6	4	7	60	11	1	1 8	0	1	1	[illegible]47	[illegible]6	06	1[illegible]8	5[illegible]8	[illegible]680	1 0	—	—	F 1 [illegible]	F g	+	+	

Appendix I Group 2 (126 to 135)

Haemodynamic and surgical data in cases having predominant mitral stenosis and mild mitral insufficiency. The group was studied by cardiac catheterization and also pressures were recorded in pulmonary artery and left auricle before and after mitral commissurotomy by direct needle puncture at the operation table

Case	Systolic, Pre	Systolic, Post	Diastolic, Pre	Diastolic, Post	Mean, Pre	Mean, Post	Arterial Oxygen Saturation	Air Ventilation Index	Oxygen Consumption Index	Arterio Venous Oxygen Difference	Cardiac Output in Litres	Cardiac Index	Stroke Volume in c c	Stroke Index	Total Pulmonary Resistance	Pulmonary Arteriolar Resistance	Mitral Resistance	Systemic Resistance	Right Ventricular Pressure Work	Left Ventricular Pressure Work	Mitral Valve Area (B) Gorlin's Formula	Surgeon: Pre	Surgeon: Post	Regurgitant Jet: Pre	Regurgitant Jet: Post	Remark
[illegible]	—	—	—	—	—	—	9	61		0 59	1	1 44	[illegible]4	1 16.1	,93	1 [illegible]8	4.8		0 64	—	—	F 3	F [illegible]	—	—	
[illegible]	[illegible]	—	1	—	4	—	[illegible]	7	1 1	1	3	1 8[illegible]	—	—	10 4	[illegible]80	644	[illegible]40	0 44	—	—	F ¼ g	F ¼	3+	+	
[illegible]	[illegible]4		16	1	[illegible]	1	[illegible]8	[illegible]6	100	94	4	1 68	44	[illegible]	8 [illegible]3	[illegible]05	19 3		0 [illegible]1	—	—	F ¼ g	F ¼ g	0	0	
[illegible]	46	—	8	—		—	6	4	161	5	5 [illegible]	83	[illegible]8	9	[illegible]8[illegible] 4 9		14	1396	0 17	—	—	Fl ¼ g	Fl ¼	0	0	
[illegible]	66	6[illegible]	[illegible]	4	1	30	0	—	—	6		[illegible]2	54	39	90	511	10	460	0 8[illegible]	—	—	F ¼ g[illegible]	F ¼ g	0	0	

Appendix I Group 3 (151 to 155)

Haemodynamic and surgical data in cases having no mitral stenosis. The group was studied by part of cardiac catheterization and was erroneously subjected to mitral commissurotomy. The absence of mitral stenosis was judged at the time of surgery

Number	Name	Age	Sex	Surface Area	History and duration of Rheumatic Heart Diseases in years	Dyspnoea: Maximum In The Past	Dyspnoea: At Admission In Hospital	Orthopnoea	Paroxysmal Nocturnal Dyspnoea	Fatigue	Haemoptysis	Oedema at Admission	Duration Of Symptoms in years	Hepatomegaly	Emboli	Functional Group (N Y Heart Association)	Auscultation: Mitral: Systolic murmur	Auscultation: Mitral: Diastolic murmur	Auscultation: Aortic	Auscultation: Pulmonary	Auscultation: Tricuspid	E C G: Rhythm	E C G: Left Auricle	E C G: Ventricle Right Or Left
301	T M	–	F	–	–	+	1+	–	–	0	–	–	–	–	–	II	0	+	0	1	0	RSR	+	[illegible]
302	J P	[illegible]	F	–	–	3+	+	–	–	0	–	–	–	–	–	II	0	+	0	P²+	0	AF + RSR	+	0
303	M J	40	F	–	–	+	1+	–	–	0	–	–	–	–	–	II	0	3+	0	P	0	AF	+	0
304	T Z	[illegible]	F	–	–	+	0	–	–	0	–	–	–	–	–	III	0	3+	0	P²+	0	AF	+	RV
305	S F	30	F	–	–	1+	0		–	0	–	–	–	–	–	III	0	+	0	P²	0	AF + RSP	+	0
306	S S	40	F	–	–	1+	1+	–	–	0	–	–	–	–	–	III	1+	3+	0	I	0	AF	+	0
307	H P		F	–	–	+	1+	–	–	0	–	–	–	–	–	III	0	+	0	P	0	AF	+	0
308	[illegible]	[illegible]	F	–	–	+	1+	–	–	1+	–	–	–	–	–	II	+	3+	0	I	0	PSR	+	0
309	D A	[illegible]	F	–	–	1+	0	–	–	0	–	–	–	–	–	II	±	1+	0	P+	0	AF	+	RV
310	K J	[illegible]	F	–	–	+	1+	–		0	–	–	–	–	–	III	0	3+	0	1 +D+	0	AF	+	[illegible]
311	S [illegible]	[illegible]	F	–	–	+	1+	–	–	0	–	–	–	–	–	III	1+	+	0	P+	0	AF	+	0
312	P	[illegible]	M	–	–	+	1+	–	–	0	–	–	–	–	–	III	0	1+	0	1+	0	ISR	+	0
313	C L	[illegible]	F	–	–	+	0	–	–	0	–	–	–	–	–	II	0	4+	0	P²	0	RSP	+	[illegible]
314	I F	[illegible]	F	–	–	1+	0	–	–	0		–	–	–	–	II	0	1+	0	1+	0	AF	+	0
315	W I	[illegible]	F	–	–	+	1+	–	–	1+	–	–	–	–	–	III	0	+	0	I	0	R R	–	0
316	W I	46	F	–	–	+	1+	–	–	±	–	–	–	–	–	III	0	3+	0	P²+	0	AF	–	0
317	V S L	41	F	–	–	1+	0	–	–		–	–	–	–	–	II	±	+	0	P²+	0	AF	+	0
318	C C	36	F	–	–	1+	1+	–	–	0	–	–	–	–	–	III	0	3+	0	P²	0	AF	+	0

Appendix III Group 7 (301 to 318)

History and clinical data in cases of pure mitral stenosis The diagnosis was made without card ac catheterisation and confirmed during mitral commissurotomy The pressures in the pulmonary artery and *left auricle* were recorded before and after commissurotomy The abbreviations used are as previously

Case	Fluoroscopy: Left Auricle	Left Ventricle	Right Ventricle	Right Auricle	Pulmonarry Artery	Mitral Valve Calc fication	Aort c Valve Calcification	Duration Of Medical Therapy: Less Than 1 Year	More Than 1 Year	Circulation Time in seconds	Venous Pressure in mm of Water	Peripheral A tery Pr ssure in mm of Hg: Systolic	Diastolic	Mean	Blood Pres ure (By Brachial Cuff)	Right Auricular Mean Pressure mm of Hg	R ght Ventricular Me n Pressure in mm of Hg	Pulmonary Arte y p essu e n mm of Hg (By Cath ter): Sy tolic	Diastolic	Mean	Pulmonary Artery Wedge Mean Pressure in mm of Hg	PAm PWm Grad ent	Iitral In ufficiency As Judged From PW Curve	Left Auricular Mean Pressure (Direct Punc ture) in mm of Hg: Pre op	Post op	Mitral Insufficiency As Judged from LA Curve: Pre op	Post op
[illegible]	3+	0	±	0	+	0	0	—	—	—	—	—	—	—	8[illegible]	—	—	—	—	—	—	—	—	6	[illegible]	0	0
[illegible]	3+	±	+	0	1+	0	0	—	—	—	—	—	—	—	6[illegible]0	—	—		—	—	—	—	—	[illegible]	[illegible]	0	0
3	3+	0	+	0	1+	+	0	—	—	—	—	—	—	—	9[illegible] 68	—	—	—	—	—	—	—	—	—	1	0	0
4	3+	[illegible]	+	0	+	0	0	—	—	—	—	—	—	—	83 6[illegible]	—	—	—	—	—	—	—	—	—	[illegible]	0	0
	3+	0	+	0	+	0	0	—	—	—	—	—	—	—	92,[illegible]	—	—	—	—	—	—	—	—	[illegible]	[illegible]	0	0
6	+	0	1+	0	1+	0	0	—	—	—	—	—	—	—	9[illegible]	—	—	—	—	—	—	—	—	[illegible]	[illegible]	0	1+
	+	0	1+	0	1+	+	0	—	—	—	—	—	—	—	11[illegible]/80	—	—	—	—	—	—	—	—	1	[illegible]	0	1+
[illegible]	+	0	+	0	+	0	0	—	—	—	—	—	—	—	1[illegible]8 8	—	—	—	—	—	—	—	—	1	0	0	0
[illegible]	+	±	+	[illegible]	+	0	0	—	—	—	—	—	—	—	10[illegible]/6[illegible]	—	—	—	—	—	—	—	—	4	[illegible]	0	0
[illegible]	+	0	+	0	+	+	[illegible]	—	—	—	—	—	—	—	1[illegible]/[illegible]8	—	—	—	—	—	—	—	—	[illegible]	[illegible]	0	0
[illegible]1	3+	0	+	0	3+	+	[illegible]	—	—	—	—	—	—	—	10 8	—	—	—	—	—	—	—	—	[illegible]	—	0	—
[illegible]	3+	0	1+	0	+	0	0	—	—	—	—	—	—	—	[illegible] 0	—	—		—	—	—	—	—	[illegible]	—	[illegible]	—
	+	0	1+	0	1+	0	0	—	—	—	—	—	—	—	110	—	—	—	—	—	—	—	—		4	0	0
[illegible]	+	0	1+	0	1+	0	0	—	—	—	—	—	—	—	8[illegible]	—	—	—	—	—	—	—	—	1[illegible]	4	—	—
[illegible]	3+	0	1+	0	+	±	0	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	[illegible]4	[illegible]	+	+
316	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	0	0
[illegible]	+	0	±	0	1+	0	0	—	—	—	—	—	—	—	10[illegible]6[illegible]	—	—	—	—	—	—	—	—	1	3[illegible]	0	0
[illegible]	+	0	+	0	1+	0	0	—	—	—	—	—	—	—	10[illegible]	—	—	—	—	—	—	—	—	[illegible]	1[illegible]	0	0

Append x III Group 7 (301 to 318)
In est gational and haemodynam c data in cases of pu e m t l sten s s The d agnos s was made w thout [illegible]rdiac catheter ati n a d conf rmed d r ng m t l comm ss rotomy The p ess res in the p lmo ary artery and left au cle were recorded befo e and after comm ssu otomy
The abbreviat ons used e as pre usly

	(By Direct Puncture) Pulmonary Artery Pressure in mm of Hg																					Mitral Valve Area As Judged By Surgeon		Regurgitant Jet Felt By The Surgeon		
	Systolic		Diastolic		Mean																					
Number	Pre Operative	Post Operative	Pre Operative	Post Operative	Pre Operative	Post Operative	Arterial Oxygen Saturation	Air Ventilation Index	Oxygen Consumption Index	Arterio Venous Oxygen Difference	Cardiac Output In Litres	Cardiac Index	Stroke Volume in c c	Stroke Index	Total Pulmonary Resistance	Pulmonary Arteriolar Resistance	Mitral Resistance	Systemic Resistance	Right Ventricular Pressure Work	Left Ventricular Pressure Work	Mitral Valve Area (By Gorlin's Formula)	Pre Operative	Post Operative	Pre Operative	Post Operative	Remarks
301	4	–	4	18	33	25	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	½ Fing r	½ Fing	0	0	
30?	48	4	8	25	[illegible]	?0	–	–		–	–	–	–	–	–	–	–	–	–	–	–	1 F ng r	1½ g r	0	0	
303	2?	–	18	18	3.	3?	–	–	–	–	–	–	–			–	–	–	–	–	–	½ Fing	Fi r	0	0	
304	6?	?	4	4	5?	50	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	½ Fi g	Fi g r	0	0	
30	41	?0	20		4	?1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	½ Fin r	½ Fi g	0	0	
306	4	4	[illegible]	[illegible]	3		–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	½ Fin r	½ Fin r	0	+	
	[illegible]	?5	[illegible]		?8	28	–	–		–	–	–	–	–	–	–		–	–	–	–	½ Fi ge	Fi g	0	0	
	?8		1	[illegible]	1	50	–	–		–	–	–	–	–	–	–	–	–	–	–	–	½ Fi g r	Fi r	0	0	
	6?	3	4	[illegible]	30	[illegible]	–		–	–	–	–	–	–	–	–	–	–	–		–	–	–	–	–	
310	6?	5?	45	4			–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	½ Fi g	1 Fi g r	0	0	
311	64	–	4		53	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	½ Fi g r	1½ Fi g r	0	0	
31	[illegible]	[illegible]	4	[illegible]	40	40	–	–	–	–	–		–	–	–		–	–	–	–	–	½ Fi g r	Fi g r	0	0	
313	6?	6.	40	40	1	?0	–	–	–	–	–	–	–	–	–	–	–		–	–	–	1 Fi g	1½ Fing r	0	0	
314	[illegible]	4	14	19	4			–	–	–	–	–	–	–	–	–	–	–	–		–	½ Fi ger	Fi g r	0	0	
315	64	5	[illegible]	4	3?	–		–	–	–	–	–	–	–	–	–	–	–	–	–	–	½ F g r	Fi g	0	0	
316	4	44	2?	?0	[illegible]	[illegible]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	½ Fi g r	Fi g r	0	0	
31	54	?8	26	40	43	4	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	½ Fi g r	Fi g r	0	0	
31?	4?	4	–	–	?8	26	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	½ Fi g	1½ F g r	0	0	

Appendix III Group 7 (301 to 318)

Haemodynamic and surgical data in cases of pure mitral stenos s The d agnos s was made without cardiac catheterizat on and confirmed dur ng mitral commissurotomy The pressures in the pulmonary artery and left auricle were recorded before and after commissurotomy

The abbreviations used are as previously

N mb	N me	Age	Sex	Surface Area	History and duration of Rheumatic Heart Diseases in years	Dyspnoea: Maximum In The Past	Dyspnoea: At Admission In Hospital	Orthopnoea	Paroxysmal Nocturnal Dy pnoea	Fatigue	Haemoptysis	Oedema at Admission	Duration Of Symptoms in years	Hepatomegaly	Emboli	Functional Group (N Y Heart Association)	Auscultati n: Mitral: Systolic murmur	Mitral: Diastolic murmur	A rtic	Pulmonary	Tricu pid	E C G: Rhythm	Left Auricle	Ventricle Right Or Left
""6	D R	41	M	—	—	+	1+	—	—	1+	—	—	—	—	—	III	3+	1+	0	P" D+	S+	A F	+	RV
3"7	I E	38	F	—	—	3+	1+	—	—	1+	—	—	—	—	—	III	1+	3+	0	P"	S+	R S R	+	RV LV
328	J J	4	F	—	—	1+	±	—	—	±	—	—	—	—	—	II	1+	+	0	P"	0	A F	+	0
329	F M	3	F	—	—	+	1+	—	—	±	—	—	—	—	—	III	1+	+	0	P I +	S+	A F	+	LV
3"0	M J	3"	M	—	—	+	1+	—	—	1+	—	—	—	—	—	II	+	3+	0	P +	0	A F	+	RV
1	A S	47	F	—	—	+	1+	—	—	1+	—	—	—	—	—	II	3+	+	0	P" D+	0	A F	+	RV
	K F	47	F	—	—	1+	±	—	—	0	—	—	—	—	—	II	0	+	0	P"	S+	A F	+	0
"T	F D	4	F	—	—	3+	0	—	—	1+	—	—	—	—	—	II	+	3+	0	P" I +	0	A F	+	RV
34	K J	46	F	—	—	+	0	—	—	0	—	—	—	—	—	II	0	+	0	P"	S+	A F	+	LV RV
"	M M	50	F	—	—	+	1+	—	—	0	—	—	—	—	—	II	±	+	0	P" +	0	A F	+	RV
"	R B	1	F	—	—	+	1+	—	—	0	—	—	—	—	—	II	1+	+	0	P +	0	A F	+	LV RV
7	I G	"	F	—	—	+	±	—	—	0	—	—	—	—	—	III	0	+	0	P I +	S+	R S R	0	RV

Append x III   Group 8 (326 to 337)

H story a d cl n cal data n cases havi g p edom nant m tral stenos s and m ld m tral nsuff c cy The d agnos s was made without card ac catheter zat on and conf rmed d r ng Comm ssu otomy The p es u es in the pulmo a y artery and left aur cle we e obta ned before and afte Comm s u ot my

"1	K S	"9	F	—	—	1+	1+	—	—	1+	—	—	—	—	—	III	1+	+	0	P" I +	S+	R S R	+	LV RV
3	M E	8	F	—	—	+	1+		—	1+	—	—	—		—	III	+	3+	0	P"	S+	A F	+	LV RV

Group 9 (351 & 352) Append x III

History and cl n cal data in cases hav ng marked m t l suff e cy The cases were e oneously subjected to m tral comm ss rotomy The p essures n the pulm na y a tery a d left a le were reco ded before and after an attempt for comm ssu otomy

The abbreviat ons used are as prev ously   L. V Left Ve t cle

Number	Fluoroscopy: Left Auricle	Fluoroscopy: Left Ventricle	Fluoroscopy: Right Ventricle	Fluoroscopy: Right Auricle	Fluoroscopy: Pulmonary Artery	Fluoroscopy: Mitral Valve Calcification	Fluoroscopy: Aortic Valve Calcification	Duration Of Medical Therapy: Less Than 1 Year	Duration Of Medical Therapy: More Than 1 Year	Circulation Time in seconds	Venous Pressure in mm of Water	Peripheral Artery in mm of Hg (By Catheter): Systolic	Peripheral Artery: Diastolic	Peripheral Artery: Mean	Blood Pressure (By Brachial Cuff)	Right Auricular Mean Pressure mm of Hg	Right Ventricular Mean Pressure in mm of Hg	Pulmonary Artery Pressure in mm of Hg (By Catheter): Systolic	Pulmonary Artery: Diastolic	Pulmonary Artery: Mean	Pulmonary Artery Wedge Mean Pressure in mm of Hg	PAm PWm Gradient	Mitral Insufficiency As Judged From PW Curve	Left Auricular Mean Pressure (Direct Puncture) in mm of Hg: Pre op	Left Auricular Mean Pressure: Post op	Mitral Insufficiency As Judged from LA ...: Pre op	Mitral Insufficiency As Judged from LA ...: Post op
3 6	4+	0	+	0	3+	+	0	–			–	–	–	–	80/ 0	–	–	–	–	–	–	–	–	3.	41	+	+
7	3+	0	+	0	+	+	0	–	–	–	–	–	–		90/[illegible]	–	–	–	–	–	–	–	–	[illegible]6	[illegible]	0	+
3 8	+	0	1+	0	1+	0	0	–	–	–	–	–	–	–	85/60	–	–	–	–	–	–	–	–	30	14	0	+
329	3+	0	1+	0	+	+	0		–	–	–	–	–	–	100/70	–	–	–	–	–	–	–	–	1	1	[illegible]	[illegible]
3 0	3+	0	1+	0	+	+	0	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	35	[illegible]	[illegible]	[illegible]
331	3+	0	+	0	+	0	0	–	–	–	–	–	–	–	10/ 0	–	–	–	–	–	–	–	–	40	[illegible]	[illegible]	[illegible]
	+	0	0	0	1+	0	0	–	–	–		–	–	–	8 /[illegible]	–	–	–	–	–	–	–	–	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	+	0	+	0	1+	+	0	–	–	–	–	–	–	–	[illegible]/70	–	–	–	–	–	–	–	–	30	2[illegible]	[illegible]	[illegible]
1	3+	1+	1+	0	2+	+	0	–	–	–	–	–	–	–	9[illegible]/63	–	–	–	–	–	–	–	–	[illegible]	36	+	+
[illegible]	3+	0	+	0	+	+	[illegible]	–	–	–	–	–	–	–	10[illegible]/	–	–	–	–	–	–		–	18	1	+	+
36	3+	1+	+	0	1+	+	0	–	–		–	–	–	–	1 0/[illegible]0	–	–	–	–	–	–	–	–	–	–	–	–
33[illegible]	1+	0	0	0	1+	0	0	–	–	–	–	–	–	–	0 / 0	–	–	–	–	–	–	–	–	–	–	–	–

Appendix III Group 8 (326 to 337)

Investigat onal and haemodynam c data in cases having predom nent mitral stenosis and m ld m tral insuff c ency The diagnosis was made without card ac catheter zat on and conf rmed during comm ssurotomy The pressures in the pulmonary artery and left aur cle were obta ned before and after commissurotomy

Number	Fluoroscopy: Left Auricle	Fluoroscopy: Left Ventricle	Fluoroscopy: Right Ventricle	Fluoroscopy: Right Auricle	Fluoroscopy: Pulmonary Artery	Fluoroscopy: Mitral Valve Calcification	Fluoroscopy: Aortic Valve Calcification	Duration Of Medical Therapy: Less Than 1 Year	Duration Of Medical Therapy: More Than 1 Year	Circulation Time in seconds	Venous Pressure in mm of Water	Peripheral Artery: Systolic	Peripheral Artery: Diastolic	Peripheral Artery: Mean	Blood Pressure (By Brachial Cuff)	Right Auricular Mean Pressure	Right Ventricular Mean Pressure	Pulmonary Artery: Systolic	Pulmonary Artery: Diastolic	Pulmonary Artery: Mean	Pulmonary Artery Wedge Mean Pressure	PAm PWm Gradient	Mitral Insufficiency As Judged From PW Curve	Left Auricular Mean Pressure: Pre op	Left Auricular Mean Pressure: Post op	Mitral Insufficiency: Pre op	Mitral Insufficiency: Post op
[illegible]1	3+	0	[illegible]+	0	+	+	0	–	–	–	–		–	–	0/60	–	–	–	–	–		–	–	31	[illegible]	+	+
[illegible]	3+	+	+	0	1+	+	0	–	–	–	–	–	–	–	110/70		–	–	–	–	–	–	–	–	–	+	+

Append x III Group 9 (351 & 352)

Invest gational and haemodynam c data in cases having marked m tr l nsuff c ency The cases were erroneously subjected to mitral commissu otomy The pressures in the pulmonary artery and left au icle were recorded before and after an attempt for commissurotomy

The abbreviations used are as previously

Numb	(By Direct Puncture) Pulmonary Artery Pressure in mm of Hg: Systolic Pre Operative	Systolic Post Operative	Diastolic Pre Operative	Diastolic Post Operative	Mean Pre Operative	Mean Post Operative	Arterial Oxygen Saturation	Air Ventilation Index	Oxygen Consumption Index	Arterio Venous Oxygen Difference	Cardiac Output in Litres	Cardiac Index	Stroke Volume in c c	Stroke Index	Total Pulmonary Resistance	Pulmonary Arteriolar Resistance	Mitral Resistance	Systemic Resistance	Right Ventricular Pressure Work	Left Ventricular Pressure Work	Mitral Valve Area (By Gorlin s Formula)	Mitral Valve Area Judged By Surgeon: Pre Operative	Post Operative	Regurgitant Jet Felt By The Surgeon: Pre Operative	Post Operative	Remarks
3	6[illegible]	–	[illegible]	–	4	[illegible]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	F ½ g	Fi ge	1+	1+	
[illegible]	5	[illegible]	30	1	40	40	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	F ½ ng	F 1½ g	+	2+	
8	0	4		[illegible]	37	3	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	+	+	
9	4		1	13	[illegible]	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	F ½ g	F 1½	+	2+	
0	63		4	[illegible]	0	[illegible]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	Fi ½ g	Fi 1½ g	1+	1+	
1	9[illegible]		68			63	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	F ½ ger	F 1 g	1+	1+	
	[illegible]	4		3	8	[illegible]	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	F ½ g	Fi 1½ ger	±	+	
[illegible]	4	48	[illegible]	6	3	40	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1+	1+	
4	50		0		38	0	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	Fi ½ g	Fi 2 g	1+	1+	
2	4		30		9	4	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	±	0	
6	5	–	3	–	4	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	Fi ½ g	Fi 2 g r	±	2+	
[illegible]		–	37	–	4	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	Fi ½ g r	Fi 1 g r	1+	1+	

Append x III Group 8 (326 to 337):

Haemody am c and su g l data n ases hav ng predom n nt m t al stenos s and *mild mitral insufficiency* The d agnos s was made w thout ca d ac cathete zat on a d conf med dur ng *comm ssurotomy* The *pressures* in the pulmona y rtery a d left au cle we e obta ned before a d after *commissu otomy*

[illegible]1	0	48	8	[illegible]	4	4	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	F 1½ g	Fi 2 g r	2+	2+	
5	50	50		–	4	40	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	F 1½ r	F 2½ r	2+		

Append x III Group 9 (351 & 352)

Haemodynam c and surg cal data n cases ha i g ma ked m t al i sufficiency The cases were [illegible] subjected to m t al comm ssu otomy The p essures the pulmonary artery and left auricle were [illegible] befo e and after an attempt for comm ssu otomy

The abb ev at ons used are as p ev ously

Number	Name	Age	Sex	Surface Area	History and duration of Rheumatic Heart Diseases in years	Dyspnoea: Maximum In The Past	Dyspnoea: At Admission In Hospital	Orthopnoea	Paroxysmal Nocturnal Dyspnoea	Fatigue	Haemoptysis	Oedema at Admission	Duration Of Symptoms in years	Hepatomegaly	Emboli	Functional Group (N Y Heart Association)	Auscultation: Mitral Systolic murmur	Auscultation: Mitral Diastolic murmur	Auscultation: Aortic	Auscultation: Pulmonary	Auscultation: Tricuspid	E C G: Rhythm	E C G: Left Auricle	E C G: Ventricle Right Or Left
1	E M	43	F	14	10	3+	1+	+	1+	1+	0	±		±	0	III	1+	1+	0	P +	S+	RSR	+	RV
02	S O	9	F	147		3+	+	+	+	1+	0	0	7	1+	0	IV	0	1+	0	D+ P+	S+	R R	+	RV
03	W E	49	F	1 7	11	4+	1+	1+	1+	0	0	0		1+	0	III	1+	1+	0	P+	0	RSR	+	RV
04	C R	43	M	–	[illegible]	+	1+	1+	0	0	±	0		1+	0	III	1+	+	0	0	0	AF	+	RV
05	C A	40	F	14		3+	+	1+	+	1+	0	0	7	1+	0	IV	0	+	0	P +	S+	AF	+	RV
06	K E	46	F	14	10	3+	+	+	+	1+	0	+		1+	1+	IV	0	1+	0	P +	S+	AF	+	RV

II Group 4 (201 to 206)

[illegible]nical data in cases having pure mitral stenosis The Group was studied by cardiac catherization [illegible] commissurotomy was performed

Number	Name	Age	Sex	Surface Area	History and duration of Rheumatic Heart Diseases in years	Dyspnoea: Maximum In The Past	Dyspnoea: At Admission In Hospital	Orthopnoea	Paroxysmal Nocturnal Dyspnoea	Fatigue	Haemoptysis	Oedema at Admission	Duration Of Symptoms in years	Hepatomegaly	Emboli	Functional Group (N Y Heart Association)	Auscultation: Mitral Systolic murmur	Auscultation: Mitral Diastolic murmur	Auscultation: Aortic	Auscultation: Pulmonary	Auscultation: Tricuspid	E C G: Rhythm	E C G: Left Auricle	E C G: Ventricle Right Or Left
	C J	4	F	11	10	1+	1+	1+	0	±	+	0	4	0	0	III		1+	0	0	S+	R R	+	RV LV
207	J L	19	F	1	8	1+	1+	1+	0	0	±	0		0	0	III	1+	+	0	P+	0	RSR	+	RV
	P R	4	F	14		3+	+	+	0	0	0	0	5	+	0	III	+	+	0	P	S+	AF	+	RV
	R B	4	M	176	1	+	1+	+	0	1+	1	0	11	0	0	III	1+	+	S+	D+	0	AF	+	LV RV
30	C M	35	F	14	[illegible]	1+	1+	0	0	1+	0	0	4	1+	0	III	+	3+	0	0	S+	AF	+	LV RV

Appendix II Group 5 (226 to 230)

History and clinical data in cases having predominant mitral stenosis and mild mitral insufficiency *The Group* was studied by cardiac catheterization and the diagnosis was confirmed at the time of commissurotomy

Number	Name	Age	Sex	Surface Area	History and duration of Rheumatic Heart Diseases in years	Dyspnoea: Maximum In The Past	Dyspnoea: At Admission In Hospital	Orthopnoea	Paroxysmal Nocturnal Dyspnoea	Fatigue	Haemoptysis	Oedema at Admission	Duration Of Symptoms in years	Hepatomegaly	Emboli	Functional Group (N Y Heart Association)	Auscultation: Mitral Systolic murmur	Auscultation: Mitral Diastolic murmur	Auscultation: Aortic	Auscultation: Pulmonary	Auscultation: Tricuspid	E C G: Rhythm	E C G: Left Auricle	E C G: Ventricle Right Or Left
221	N A	6	M	1	9	1+	1+	1+	0	1+	±	0		2+	1+	IV	+	3+	±	0	0	AF	+	LV RV

Appendix II Group 6 (251)

History and clinical data in a case having *marked* mitral insufficiency The case was erroneously *subjected* to mitral commissurotomy when the diagnosis was *confirmed*

	Fluoroscopy							Duration Of Medical Therapy				Peripheral Artery in mm of Hg (By Catheter)						Pulmonary Artery Pressure in mm of Hg (By Catheter)						Left Auricular Mean Pressure (Direct Puncture) in mm of Hg		Mitral Insufficiency As Judged from LA Curve	
[illegible]	Left Auricle	Left Ventricle	Right Ventricle	Right Auricle	Pulmonary Artery	Mitral Valve Calcification	Aortic Valve Calcification	Less Than 1 Year	More Than 1 Year	Circulation Time in seconds	Venous Pressure in mm of Water	Systolic	Diastolic	Mean	Blood Pressure (By Brachial Cuff)	Right Auricular Mean Pressure mm of Hg	Right Ventricular Mean Pressure in mm of Hg	Systolic	Diastolic	Mean	Pulmonary Artery Wedge Mean Pressure in mm of Hg	PAm PWm Gradient	Mitral Insufficiency As Judged From PW Curve	Pre op	Post op	Pre op	Post op
1	+	0	+	0	+	0	0	—	+	11	160	1	0	91			[illegible]	1	[illegible]	4	3	0	0	—	—	—	—
	—	—	—	—	—	—	—	—	+	0	103	9	54	0	—	—	—	1			19	9	0	—	—	—	—
	+	0	+	0	+	0	0	—	+	[illegible]	210	98	4		—	—	40	[illegible]	4	67	—	—		—	—	—	—
	3+	1+	+	1+	+	+	0	—	+	11	30	—	—	—	115[illegible]	11	0			4	—	—	—	—	—	—	—
	3+	0	+	1+	3+	0	0	—	+	4	50	94	1		—	6	6	91	83	61	4	[illegible]	+	—	—	—	—
	+	0	1+	0	+	0	0	—	+	16	110	11	1	11	—		11	1	14	19	11	6	0	—	—	—	—

pend x II Group 4 (201 to 206)

est gat onal and haemodynamic data n cases hav ng pu e mit al stenos s The g oup was st d ed by ca d ac heterizat on and mitral commissurotomy was pe formed

	1+	1+	1+	1+	1+	0	0	—	+	18	101	14	90	10	—		11		[illegible]	8	11	17	0	—	—	—	—
	+	0	+	[illegible]	1+	0	0	—	+	19	60	—		—	—		30		40		—	—		—	—		—
	3+	±	+	+	+	+	0	—	+	—	—		—	—		—	—	—	—	—	—	—	—	—	—	—	—
	+	1+	+	1+	1+	0	[illegible]	—	+	—	[illegible]0	1	7	8		10	0	4		0	1	1	0	—	—	—	—
	3+	1+	+	1+	+	0	0	—	+	1	0		4	9				41	4	18	8	10		—	—	—	—

pend x II G oup 5 (226 to 230)

estigational and haemodynamic data n cases having p edom n nt m t al stenos s and m ld m t al suff – ncy The Group was stud ed by ca d ac catheterza t on and the d agnos s was conf rmed at the t me of nm ssu otomy

	+	1+	+	1+		±	0	—	+	4	[illegible]0	217	0	8[illegible]	—		30	68	31	41	—	—	—	—	—	—	—

ppend x II Group 6 (251)

vest gat onal and haemodynamic d ta in a case hav ng marked m tral i s ff c ency The case was erroneously ubjected to mitral commissurotomy when the d agnos s was conf rmed

Number	(By Direct Puncture) Pulmonary Artery Pressure in mm of Hg — Systolic — Pre Operative	Systolic — Post Operative	Diastolic — Pre Operative	Diastolic — Post Operative	Mean — Pre Operative	Mean — Post Operative	Arterial Oxygen Saturation	Air Ventilation Index	Oxygen Consumption Index	Arterio Venous Oxygen Difference	Cardiac Output In Litres	Cardiac Index	Stroke Volume in c c	Stroke Index	Total Pulmonary Resistance	Pulmonary Arteriolar Resistance	M tral Re istance	Systemic Resistance	Right Ventricular Pressure Work	Left Ventricular Pressure Work	M tral Valve Area (By Gorl n s Formula)	Mitral Valve Area As Judged By Surgeon — Pre Operative	Mitral Valve Area As Judged By Surgeon — Po t Operative	Regurgitant Jet Felt By The Surgeon — Pre Ope ative	Regurgitant Jet Felt By The Surgeon — Po Op at ve	R marks
01	–	–	–	–	–	–			14	16	41(	61			8	8'	144	1 18 1	–	1 1	F	1	1¼ Fi g r	0	0	t
0?	–	–	–	–	–	–	91	3 08	11	41)	3 8	61	30	61		08 4		110 0 88	–	0 8	F	1	1¼ Fi r	0	0	
0	–	–	–	–	–	–	–	3 J	1	5 80	4 6			1 1 18	1 6		J	11 6 1 90	–	0 r	F	1	1¼ F g r	0	0	
04	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	¼ g	Ff e	0	+	
	–	–	–	–	–	–	91	4 61	14	J	0	1 8	0 14	1 0		48	1 77	1 1	–	0 1	Fi	½	1¼ F r	0	0	h
06	–	–	–	–	–	–	88	3 13	11	6 10	0	1 11	–	–	6		8 8	740	–	1 1	–	–	–	–	–	P

ll Group 4 (201 to 206)

... and surg cal data in cases having pure mitral stenos s The group was studied by ... iac catheterizat on and mitral comm ssurotomy was performed

Number																										
???	–	–	–	–	–	–	F	a	1	41	(	41	8	4 1	01		10 1 0		–	–	Fi ger	1	F g	+	–	
???	–	–	–	–	–	–	–	40	11	4 40	4 8		0 18	1 10	–	–	–	)	–	–	Fi g r	¼	F	+	+	
2?3	–	–	–	–	–	–	–		18	8 1	J	0	0	d	–	–	–	–	–	–	F g	¼	Fi r	+	+	
??9	–	–	–	–	–	–	9?	?)	8	8 0	1 8	1 0	1	1 00		0 010	4100	0	–	–	F r	1	1¼ Fi r	3+	+	
2?0	–	–	–	–	–	–	3)	8 1		0 6	8	1 10	–	–	10 8		8	80 164	0 9	–	–	Fi 1	1¼ Fi 1	3+	+	

Appendix ll Group 5 (226 to 230)

Haemodynam c and surg cal data in cases having predominent mitral stenosis and m ld mitral i suff ciency The group was studied by card ac catheterizat on and the diagnosis was confirmed at the time of commissurotomy

Number																										
251	–	–	–	–	–	–	14	3	11	(1		1 8	–	–	9 0	–	–	110 0	–	–	F	1	–	1+	1+	

Apppendix ll Group 6 (251)

Haemodynamic and surg cal data n a case hav ng ma ktd m t al nsuff c ency The case was erroneously subjected to miral commissu otomy when the diagnos s was confirmed

Number	Name	Age	Sex	Surface Area	History and duration of Rheumatic Heart Disease in years	Dyspnoea: Maximum In The Past	Dyspnoea: At Admission In Hospital	Orthopnoea	Paroxysmal Nocturnal Dyspnoea	Fatigue	Haemoptysis	Oedema at Admission	Duration Of Symptoms in years	Hepatomegaly	Emboli	Functional Group (N Y Heart Association)	Auscultation: Mitral: Systolic murmur	Auscultation: Mitral: Diastolic murmur	Auscultation: Aortic	Auscultation: Pulmonary	Auscultation: Tricuspid	E C G: Rhythm	E C G: Left Auricle	E C G: Ventricle Right Or Left
11	W	–	M	1 44		+	1+	1+	0	)	1+	+	1		0	III	+	+	I +	D+ P+	S+	RSR	+	RV + LV +
4	Y J	–	M	1 64	4	1+	1+	0	0	±	0	1+	3	1	0	III	+	1+	S±	1 +	S+	AF	+	RV + LV1+
4 1	N C		F	1 1	10	1+	1+	0	0	+	1+	+	1)	3	1+	II	+	1+	0	I + P+	S+	AF	+	PV +
14	S K	3	M	1 64	16	1+	1+	1+	1+	+	1+	0		±	0	III	4+	+	S+	I +	0	RSR	+	LVI+
60	S	2	F	1 1		1+	1–	0	0	1+	0	(		1	1+	III	4+	+	0	I +	S+	RSI	+	LV +
06	P M	–	F	1 4		1+	1+	1+	0	1+	1+	)	3	0	0	II	+	+	0	1 +	S+	RSR	+	LV+ RV +
0	I R	19	M	19	6	1+	1+	0	0	(	+	0		0	0	II	+	3+	D+	0	0	AF	+	0
08	K Y	Y	F	1 9	)	+	1	1+	±	+	0	1+		3+	+	III	4+	+	0	I +	S+	R R	+	LV +
09	C M	–	F	1..	?	+	1+	1+	0	0	1+	1+		3+	0	III	)	+	S+	P+	0	AF	+	RV + LV +
61	W S	61	F	1 4	40	+	1	+	0	1+		1+	)	+	0	III	+	1+	I + S+	P +	0	AF	+	LV +
61	M (		F	1 4	8	+	1+	1+	1+	1+	0	+		4+	+	IV	1+	+		I +	S+	R R	+	RV3+
	S 1	.	F	1.5	18	+	1–	0	0	–	3	+		1+		II	+	+	S+	I +	+	P R	+	RV + LV1+
63	R J	–	M	1 1	)	1+	±	J	0	1+	0		1 1			II	3+	1+	0	I +	0	AF R R	+	)

Appe d x IV G up 10 (451 t 463)

H sto y and cl n cal data in cases ha ing p edom n nt m t al insuff ci ncy Th se we e stud ed by card ac cathet at on nd ejected fo m t al comm ssu tomy becau e of m t l nsuff en y

Number	Name	Age	Sex	Surface Area	History and duration of Rheumatic Heart Disease in years	Dyspnoea: Maximum In The Past	Dyspnoea: At Admission In Hospital	Orthopnoea	Paroxysmal Nocturnal Dyspnoea	Fatigue	Haemoptysis	Oedema at Admission	Duration Of Symptoms in years	Hepatomegaly	Emboli	Functional Group (N Y Heart Association)	Auscultation: Mitral: Systolic murmur	Auscultation: Mitral: Diastolic murmur	Auscultation: Aortic	Auscultation: Pulmonary	Auscultation: Tricuspid	E C G: Rhythm	E C G: Left Auricle	E C G: Ventricle Right Or Left
01	M P		M	1 4		+	1+	1+	0	±		1+	4	+	1+	III	+	+	S+	I +	S+	AF	+	RV+
	R F	41	M	1	1	1+	1+	1+	0	1+	1+	+		1+	0	III	0	+	0	1 +	S+	AF	+	RV+
03	I 1		F	1	4	1+	1+	)	0	0	0	0		0	(	I	0	+	0	0	0	R R	+	RV±
04	N I	4	F	1	1	+	±	0	1+		0	)	1	1+	0	I	1+	1+	0	0	0	R R	+	RV+
4	I I	40	F	1 44		1+	±	0	+	0	0	0	"	1+	0	II	1+	+	S+	0	0	P R	+	RV+
06	B B	41	F	1 1		1+	±	+	0	+	0	+	4	+	+	II	1+	+	±	P+		AF	+	RV+

Number	Fluoroscopy: Left Auricle	Fluoroscopy: Left Ventricle	Fluoroscopy: Right Ventricle	Fluoroscopy: Right Auricle	Fluoroscopy: Pulmonary Artery	Fluoroscopy: Mitral Valve Calcification	Fluoroscopy: Aortic Valve Calcification	Duration Of Medical Therapy: Less Than 1 Year	Duration Of Medical Therapy: More Than 1 Year	Circulation Time in seconds	Venous Pressure in mm of Water	Peripheral Artery Pressure in mm of Hg: Systolic	Peripheral Artery Pressure in mm of Hg: Diastolic	Peripheral Artery Pressure in mm of Hg: Mean	Blood Pressure (By Brachial Cuff)	Right Auricular Mean Pressure mm of Hg	Right Ventricular Mean Pressure in mm of Hg	Pulmonary Artery pressure in mm of Hg (By Catheter): Systolic	Pulmonary Artery pressure in mm of Hg (By Catheter): Diastolic	Pulmonary Artery pressure in mm of Hg (By Catheter): Mean	Pulmonary Artery Wedge Mean Pressure in mm of Hg	PAm PWm Gradient	Mitral Insufficiency As Judged From PW Curve	Left Auricular Mean Pressure (Direct Puncture) in mm of Hg: Pre op	Left Auricular Mean Pressure (Direct Puncture) in mm of Hg: Post-op	Mitral Insufficiency As Judged from LA Curve: Pre op	Mitral Insufficiency As Judged from LA Curve: Post op
451	3+	+	+	+	+	1+	0	+	–	3	1	1	0	8	–		1	90	4	6	8	3	+	–	–	–	–
4[illegible]	+	+	+	0	1+	+	0	–	+	1		1 8	81	8	–	6	17		5	41	4	17	–	–	–	–	–
4 3	+	+	4+	1+	1+	0	0	–	+	4	1	11	88	9	–	16	49	104		61	0	31	–	–	–	–	–
454	1+	+	1+	+	1+	0	+	–	+		1	118	48	89	–	–	[illegible]	30	4	8	1	4	0	–	–	–	–
455	1+	[illegible]	1+	0	1+	+	0	+	–		101	11	71		–	5	16	31	11	1	1	9	+	–	–	–	–
456	+	1+	+	0	1+	0	0	+	–		10	11[illegible]	6[illegible]	8	–	1	33		43	4	[illegible]	18	0	–	–	–	–
[illegible]		±	1+	+	+	±	0	–	+	1	170	11	5	9	–	6	16	1	14	17	1	4	+	–	–	–	–
45[illegible]	3	±	+	1+	1+	0	0	–	+	0		1[illegible]	6	9	–	13	43	11	1	6	–	–	–	–	–	–	–
[illegible]	3+	+	+	+	3+	0	0	–	+	49	100	–	–		–	–	–	–	–		[illegible]	20	–	–	–	–	–
460	4+	+	+	1+	+	+	+	–	+	4	90	1 9	6	0	180/[illegible]0	[illegible]	13	5	[illegible]	7	10	[illegible]	+	–	–	–	–
461	+	+	+	+	+	+	0	–	+	–	4	11	6	8	90 60		50	1	5	8	–	–	–	–	–	–	–
462	[illegible]+	1+	1+	1+	0	0	0	–	+	9	9	11	71	8	1[illegible]/75	6	1	[illegible]	17	[illegible]	5	1	–	–	–	–	–
46[illegible]	1+	±	0	0	0	0	0	+	–	8	98	–	+	–	–	–	–	–	–	–	–	–	–	–	–	–	–

Append x IV Group 10 (451 to 463)

Investigational and haemodynamic data in cases having predominant m tral insuff ciency The cases were stud ed by ca diac catheterizat on and rejected for mitral comm ssurotomy because of m tral insuff ciency

401	4+	1+	3+	+	3+	+	0	+	–	17	1[illegible]0	90	80	8[illegible]	110/90	9	3[illegible]	7	53	60	–	–	–	–	–	–	–
402	3+	±	1+	1+	+	0	0	–	+	[illegible]6	1[illegible]0	1[illegible]	60		1 0/90	3	1	8	20	–	–	–	–	–	–	–	–
403	+	0	1+	0	±	0	0	+	–	[illegible]0	60	10	[illegible]	80	110/[illegible]0	0	10	31	10	1	10	7	0	–	–	–	–
404	1+	0	1+	0	0	0	0	+	–	14	0	103	[illegible]0	6	11[illegible]0	3	1	[illegible]6	10	22	18	6	0	–	–	–	–
405	1+	0	1+	0	1+	0	0	–	+	13	100	12[illegible]	3	[illegible]	–	8	14	[illegible]	10	1	11	4	–	–	–	–	–
406	4+	±	1+	1+	1+	+	0	–	+	–	–	1[illegible]0	7	100	–	7	18	[illegible]0		[illegible]0	–	–	–	–	–	–	–

Name	(By Direct Puncture) Pulmonary Artery Pressure in mm of Hg						Arterial Oxygen Saturation	Air Ventilation Index	Oxygen Consumption Index	Arterio Venous Oxygen Difference	Cardiac Output In Litres	Cardiac Index	Stroke Volume in c c	Stroke Ind x	Total Pulmonary Resistance	Pulmonary Arteriolar Re istance	Mitral Resistance	Systemic Res stance	Right Ventricular Pres ure Work	Left Ventricular Pressure Work	Mitral Valve Area (By Gorlin s Formula)	Mitral Valve Area As Judged By Surgeon		Regurgitant Jet Felt By The Surgeon		Remarks
	Systolic		Diastolic		Mean																					
	P e Op tive	Post Oper tiv	Pre Op r tive	Post Oper tive	Pre Operative	Post Operative																Pre Operative	Post Ope ative	Pre Operative	Post Operat ve	
1	—	—	—	—	—	—	96		1 3	81	4	3	33	3	1 9	31	604	193	1	—	—	—	—	—	—	
	—	—	—	—	—	—	91	1	1	6 4	3 1	01	7	5	9"6	40	1	1 0	0 91	—	—	—	—	—	—	
3	—	—	—	—	—	—	—	4 18	1 1	9 81		1 7	11	10	740	139	131	44 0	0 1	—	—	—	—	—	—	
	—	—	—	—	—	—	—	30	13	5 49	1	7	3	—	30	6	4 1	1 40	—	—	—	—	—	—	—	
	—	—	—	—	—	—	100	3 88	171	4	6 4 6	04	8	34	413	1 5	4	130	0 61	—	—		—	—	—	
	—	—	—	—	—	—	—	3 09	1	6	5	1 83		16	1 13	6 1		1 0	—	—	—	—	—	—	—	
	—	—	—	—		—	9		89	13	4	1 18	4	1	6	9	461	0	0	—	—	—	—	—	—	
	—	—	—	—	—	—	—	10	143	10 3	1 7	1 6	14	10	3	1434	037	4 6	0 9	—	—		—	—	—	
	—	—	—	—	—	—	91	60	117	4 5	9 9	6	61	40	1418	40	1013	1 0	1 3	—	—	—	—	—	—	
0	—	—	—	—	—	—	—	4	1	7 6	9	1 37		3	7	01	4	0	0 7	—	—	—	—	—	—	
1	—	—	—	—	—	—	—	6 1	140	9	1	1 4	5	11	3 10	—	—	31	1	—	—	—	—	—	—	
	—	—	—	—	—	—	97	—	14	6 9 5		15	4	1	6"4		433	116	0 61	—	—	—	—	—	—	
3	—	—	—	—	—	—	9	9	11		34 6	0	—	—	—	—	—	—	—	—	—	—	—	—	—	

Append IV G oup 10 (451 to 463)

Hæmodynam c data n cases hav ng p edom nant m tral nsuff ciency The cases were stud ed by Cardiac Cathete izat on and rejected for M tral Comm ssurotomy because of m tral insuff c ency

401	—	—	—	—	—	—	—	4 86	130	9.85	3	1.4	6	15	330	—	—	0	1 19	—	—	—	—	—	—	
40	—	—	—	—	—	—	—	4 6	1 1	7.37	96	1	6	—	—	—	—	3	0 51	—	—	—	—	—	—	
403	—	—	—	—	—	—	—	98		4 2	4 61	0	8	28	290	119	1 1	136	0	—	1 63	—	—	—	—	
404	—	—	—	—	—	—	96	91	119	5 1	3 1	34	—	—	68	296		16"8	0 41	—	—	—	—	—	—	
405	—	—	—	—	—	—	—	3	108	4 11	3 6	6	47	33	319	106	1	1 00	0 6	—	1 3	—	—	—	—	
406	—	—	—	—	—	—	—	4 1		4 63	58	6	41	313	10	41	1		0 48	—	—	—	—	—	—	

						Dyspnoea											Auscultation					E C G		
																	Mitral							
Number	Name	Age	Sex	Surface Area	History and duration of Rheumatic Heart Diseases in years	Maximum In The Past	At Admission In Hospital	Orthopnoea	Paroxysmal Nocturnal Dyspnoea	Fatigue	Haemoptysis	Oedema at Admission	Duration Of Symptoms in years	Hepatomegaly	Emboli	Functional Group (N Y Heart Association)	Systolic murmur	Diastolic murmur	Aortic	Pulmonary	Tricuspid	Rhythm	Left Auricle	Ventricle Right Or Left
40?	T G	~	F	1 ?9	14	+	1+	0	0	0	0	±	1	0	0	II	2+	+	0	P²+	S+	P I	+	RV+ LV+
408	R J	?0	M	1 8?	6	+	1+	0	1+	0	±	0	6	0	0	I	0	+	0	P²+	0	I SI	+	RV+
409	H R	~	F	1	11	+	1+	0	±	0	0	0		0	0	II	0	+	0	P²+	0	AF	−	RV+
410	M C	0	F	1.0?		+	1+	0	±	0	1+	0	4	0	0	II	+	+	[illegible]	D+	S+	R P	+	RV+
411	C J	?8	F	1 6	14	±	0	0	0	0	0	0	14	0	0	I	±	1+	0	[illegible]	0	RSR	0	0
41?	W F	?8	F	1.?1	?0	1+	±	±	0	±	0	0	1	1+	0	I	0	1+		P+	0	RSI	+	RV±
413	L B	41	F	1 46	11	1+	±	0	0	0		0		+	0	II	1+	+	0	P²+	0	−	+	LV+ RV+
414	W H	41	M	1.8	14	+	1+	0	1+	0	[illegible]	1	14	±	0	I	[illegible]	1+	0	0	0	R B	+	RV+
[illegible]	[illegible]	[illegible]	F	1 ?9	4	1+	0	1+	0	0	±	1+	10	±	0	I	+	1+	0	I +	0	AF	+	0
[illegible]	[illegible]	[illegible]	F	1.08	10	+	1+	+	0	+	[illegible]	+	10	+	0	II	+	?+	0	I + I +	S+	AF	+	RV+ LV+
[illegible]	? R	47	F	1.6?	8	+	1+	[illegible]	[illegible]	[illegible]	0	[illegible]	1	0	0	II	0	1+	[illegible]	I +	[illegible]	RSI	+	0
418	S S	?3	F	1.4?	2?	+	±	0	0	0	0		14	±	0	II	+	1+	0	I +	S+	RSI AF	+	RV+
419	G R	4?	F	1 28	1	2+	1+	1+	0	1+	+	+	17	2+	0	IV	+	+	0	P²+	S+	RSR AF	+	LV+ RV+
420	H M	32	F	1 ?4	0	2+	1+	+	0	1+	1+	+		+	0	IV	+	+	0	I + I +	S+	RSR	+	RV+ LV+
421	M ?	2?	F	1 ??		2+	1+	+	0	1+	0	+	6	+	0	III	+	1+	0	P²+	S+	ISI	+	LV+ LV+
422	? ?	?	M	1 6?	6	2+	1+	1+	0	1+	0	+		1+	1+	III	3+	+	S+	I +	S+	R R	+	LV+ LV+
423	D M	41	M	1	1	1+	±	1	0	±		1	1	1+	0	II	+	1+	S+	P²+	0	RSR	+	RV+ LV+
424	Z D	4?	M	1 6?	4	+	1+	−		0	0	0		0	0	I	0	1+	S+	0	0	R R	+	RV+ LV+
425	D P	??	F	1.??	1	1+	±	[illegible]	0	0	1+	0		0	0	I	+	1+	D+ S+	0	[illegible]	I R	+	RV+ LV±

Appendix IV Group II (407 to 425)

History and clinical data in cases suspected of predominant mitral stenosis. The cases were studied by cardiac catheterization and rejected for mitral commissurotomy for reasons other than mitral insufficiency.

Nu	Fluoroscopy							Duration Of Medical Therapy		Circulation Time in seconds	Venous Pres ure in mm of Water	Peripheral Artery in mm of Hg (By Catheter)			Blood Pressure (By Brachial Cuff)	Right Auriculr Mean Pres ure mm of Hg	Right Ventricular Mean Pressure in mm of Hg	Pulmonary Artery Pressu e in mm of Hg (By Catheter)			Pulmonary Artery Wedge Mean Pressure in mm of Hg	PAm PWm Gradient	Mitral In ifficiency As Judged From PW Curve	Left Auricular Mean Pressure (Direct Puncture) in mm of Hg		Mitral Insufficiency As Judged from LA Curve	
	L ft Au icle	Left Ventricle	Right Ventricle	Right Auricle	Pulmonary Artery	Mitral Valve Calcification	Aortic Valve Calcification	Less Than 1 1ear	More Than 1 Year			S stolic	Diastolic	Mean				S tolic	Diastolic	Mean				Pre op	Post op	Pre op	Post op
40	1+	±	±	0	±	0	0	–	+	14	90	118	0	8	–		1	40	0			6	8	–	–	–	–
408	+	0	1+	±	+	+	0	+	–	1	4	12		10	–	9	20	4 (	3	20			0	–	–	–	–
409	+	0	1+	0	1+	+	0	–	+	–	–	10.	50	0	–		18	4	20	2	99		0	–	–	–	–
410	1+	0	1+	0	1+	+	0	+	–	–	–	101	65	6	1 0 85	9		46		2	4		0	–	–	–	–
411	–	–	–	–	–	–	–	+	–	–	110	110	63	81	–	8	1	–	–	–	–	–	–	–	–	–	–
41	1+	0	±	0	±	0	0	+	–	11	90	140	–	9	–	6	11		10	1	–	–	–	–	–	–	–
4 3	3+	1+	+	1+	+	±	(	+	–	1	100	8	1	7	–	–	19	40	10	4	–	–	–	–	–	–	–
414	+	0	1+	±	1+	±	–	–	+	14	100	1			–	1	11	29	13	0	3		0	–	–	–	–
41	1+	0	0	0	0	0		–	+	–	–	11	60	4	–		1	19	10	1	10	3	3	–	–	–	–
416	+	1+	3+	0	+	0	0	–	+	3	10	90	61	4	–		8		1	1			+	–	–	–	–
41	1+	0	0	0	0	(	0	–	+	1	4	–	–	90	–	6	1	44	1	6		11	0	–	–	–	–
418	+	0	+	1+	+	0	0	+	–	1	179	90	6		–	1	41	0	9	41	–3	16	0	–	–	–	–
419	4+	1+	+	3+	1+	0	0	–	+	96	90	115	6.	85	–	6	60	11	~	6	37	28	–	–	–	–	–
4 0	+	+	+	+	+	0	0	–	+	5	38	114	4	80	–			80	1	4	–	–	–	–	–	–	–
4 1	4+	1+	+	1+	+	0	0	–	+	40		1 1 1	8	140	–	11		148	0	8	4	40	+	–	–	–	–
42	+	1+	+	3+	+	+	)	–	+	33	1	104	0	0	–	10	4	113	4	0	–	–	–	–	–	–	–
4 3	+	1+	1+	±	1+	0	0	+	–	96	6	114		64	–	3	11	58	18	~	6	71	– 0	–	–	–	–
4 4	1+	+	1+	1+	1+	0	+	–	–	14	3	3	8		110 60	6	1	4		34	19	1	–	–	–	–	–
4	1+	±	1+	0	1+	0	0	–	+	–	–	96	60	64	–	4	9	10		1			0	–	–	–	–

Append x IV G o p 11 (401 to 425)

Invest gat on 1 a d haemodynom c d ta n ca s susp cted of p edom nant m t al stenos s The cases we e stud ed by c rd ac cathete zat on and ject d f m t al omm ssurotomy for re so s othe th n m tral n-s ff c ency

Number	Pulmonary Artery Pressure in mm of Hg (By Direct Puncture) Systolic Pre Operative	Systolic Post Operative	Diastolic Pre Operative	Diastolic Post Operative	Mean Pre Operative	Mean Post Operative	Arterial Oxygen Saturation	Air Ventilation Index	Oxygen Consumption Index	Arterio Venous Oxygen Difference	Cardiac Output in Litres	Cardiac Index	Stroke Volume in c c	Stroke Index	Total Pulmonary Resistance	Pulmonary Arteriolar Resistance	Mitral Resistance	Systemic Resistance	Right Ventricular Pressure Work	Left Ventricular Pressure Work	Mitral Valve Area (By Gorlin's Formula)	Mitral Valve Area As Judged By Surgeon Pre Operative	Mitral Valve Area As Judged By Surgeon Post Operative	Regurgitant Jet Felt By The Surgeon Pre Operative	Regurgitant Jet Felt By The Surgeon Post Operative	Remark
407	—	—	—	—	—	—	93	3 53	1[illegible]	3 6	5 8[illegible]	4.[illegible]	5[illegible]	4	[illegible]	81	30[illegible]	118[illegible]	1 4	—	—	—	—	—	—	
408	—	—	—		—	—	96	51	111	4 43	4 8	5	64	1	0[illegible]	1[illegible]	38	1 46	—	—	0 8	—	—	—	—	
409	—	—	—	—	—	—	97	5 07	1[illegible]	4 55	4.2[illegible]	68	[illegible]	[illegible]	509	1	41	1328	0.90	—	0 8[illegible]	—	—	—	—	
410	—	—	—	—	—	—	98	3 5	143	4 3	5.51	[illegible]8	60	4	[illegible]	[illegible]07	[illegible]	110[illegible]	1 41	—	0 8[illegible]	—	—	—	—	
411	—	—	—	—	—	—	—	4	153	4 08	6 0	3	5	34	—	—	—	—	—	—	—	—	—	—	—	
41[illegible]	—	—	—	—	—	—	91	60	111	4 1	4 01	66	0	3	4[illegible]	—	—	18[illegible]	0[illegible]	—	—	—	—	—	—	
413	—	—	—	—	—	—	94	3 74	1[illegible]7	41	51	1 7	4	[illegible]	65	320	445	4 8	0[illegible]	—	—	—	—	—	—	
414	—	—	—	—	—	—	93	3.98	130	4 03	6	3.46	56	49		63	194	9[illegible]	—	—	—	—	—	—	—	
[illegible]		—	—	—	—	—	97	3 03	116	4 9[illegible]	3.[illegible]3	[illegible]	34	24	32[illegible]	74	48	10[illegible]7	0 4	—	1 1	—	—	—	—	
[illegible]		—	—	—	—	—	9	6	98	6 0[illegible]	2 6[illegible]	1.0[illegible]	[illegible]	24	51	41	[illegible]71	2230	0 3	—	1 0	—	—	—	—	
	—	—	—	—	—	—	95	6	11	3.5	3 2	3 13	43	[illegible]6	416	1[illegible]	[illegible]	[illegible]	—	—	0 5	—	—	—	—	
418	—	—	—	—	—	—	97	4 0	103	5 45	4.3[illegible]	3 [illegible]	41	3[illegible]	54	[illegible]04	460	1610	—	—	0 8	—	—	—	—	
419	—	—	—	—	—		—	5 3	136	8 3	1	1 64	14	11	860	1430	1430	3[illegible]40	—	—	—	—	—	—	—	
4[illegible]0	—	—	—	—	—	—	—	5 4	1 8	7 14	3	1 60		19	16[illegible]0	—	—	8 0	—	—	—	—	—	—	—	
4[illegible]1	—	—	—	—	—	—	82	3 6	113	9 3	1.8	1 16	13	8	[illegible]40	1 80	2[illegible]	4410	1 16	—	—	—	—	—	—	
4[illegible]	—	—	—	—	—	—	83	3 87	13[illegible]	10 4	14	1 8	4	14	[illegible]0	—		840	1 1	—	—	—	—	—	—	
4[illegible]3	—	—	—	—	—	—	98	3 58	1 4	3 4	3 05	3	44	33	46	4[illegible]	1 1	11	0 3	—	—	—	—	—	—	
4[illegible]4	—	—	—	—	—	—	96	93	1 8	3 05	4 45	3	[illegible]	23	610	—		1346	1 14	—	—	—	—	—	—	
4[illegible]	—	—	—	—	—	—	—	18	11	4 08	4 4	8[illegible]		[illegible]	[illegible]00		1[illegible]8	1155	0[illegible]	—	1	—	—	—	—	

Appendix IV Group II (401 to 425)

Haemodynamic data in cases suspected of predominant mitral stenosis The cases were studied by cardiac catheterization and rejected for mitral commissurotomy for reasons other than mitral insufficiency

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